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BOLETIN

DE LA

ASOCIACION MEDICA DE PUERTO RICO

VOL. 50 ENERO 1958 NO. 1

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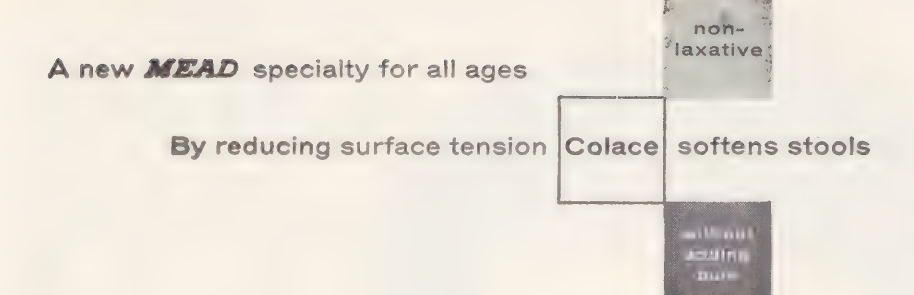
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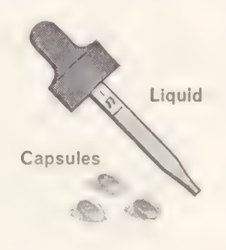
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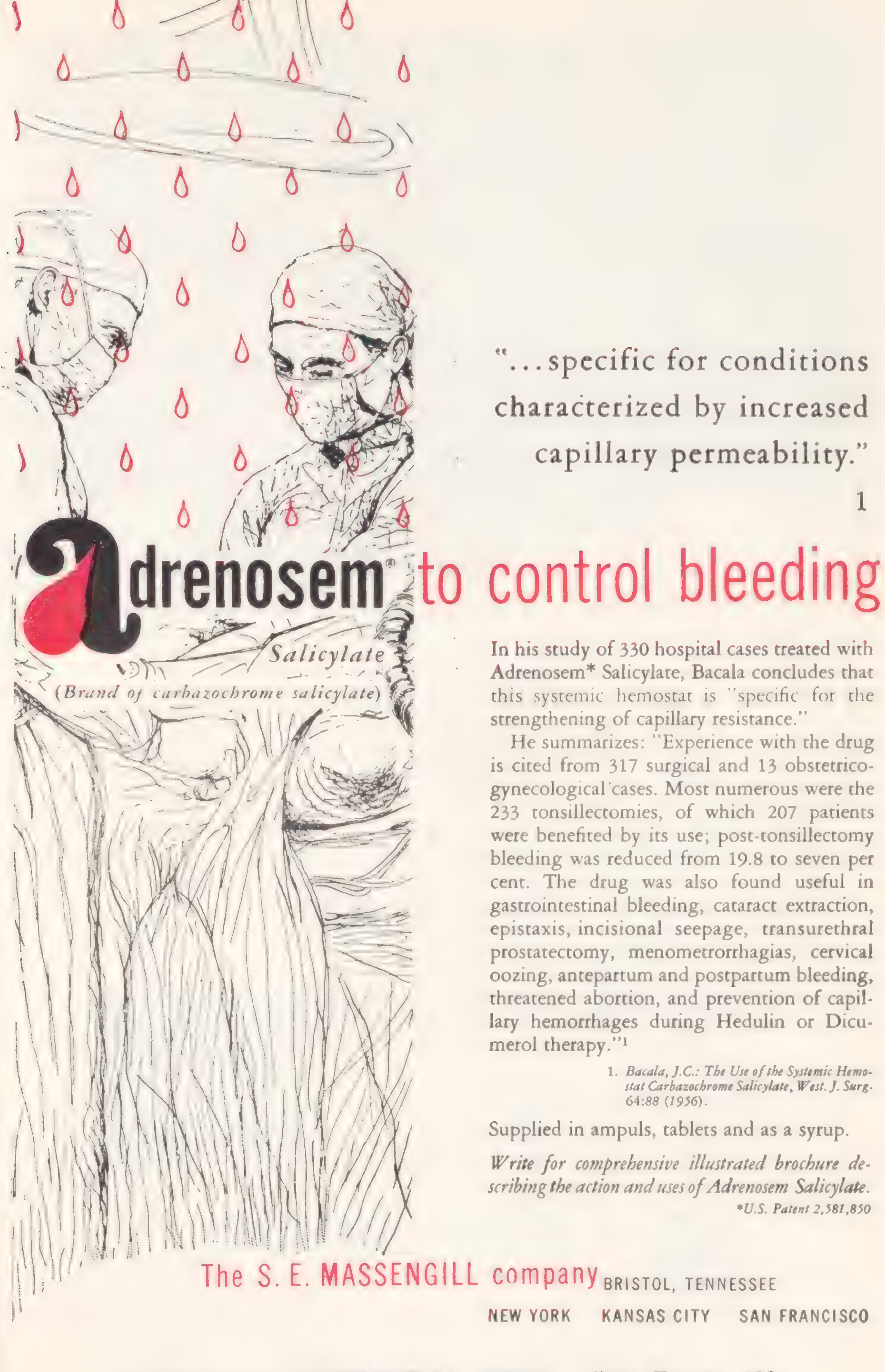
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DE LA ASOCIACION MEDICA DE PUERTO RICO

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STUDIES ON BLOOD COPPER IN NORMAL AND PATHOLOGICAL STATES*

*RAMON M. SUÁREZ, HORTENSIA F. DE BERROCAL, SARA T. DE OLAVARRIETA, RAMON M. SUÁREZ JR., ROBERTO BUSÓ and JUAN SABATER***

Copper is present in water, soil, plant and animal cells, etc. The richest sources of copper are in descending order of concentration, nuts, dried legumes, cereals, dried fruits, poultry, fish, animal tissues, green legumes, roots and tubers, leafy vegetables such as cauliflower and celery, fresh fruits and non-leafy vegetables. Several of these foodstuffs are rarely if ever consumed by the native Puerto Rican.

Copper forms an essential element of the molecule of **hemocyanin,**[1] which is the blue pigment, functionally similar to hemoglobin, found in mollusks and in other marine animals and also of **turacin,** which is the brilliant red-colored pigment found in the feathers of certain birds, especially in some African species. In man nearly 96 per cent of all serum copper is found in the form of a blue copper-globulin complex known as **ceruloplasmin.**

Although the full biological significance of copper in man remains unknown or controversial, its deficiency in animals have been definitely proven. The "falling disease" of cattle in Australia and the "swayback" in newborn and young lambs in Australia and Great Britain have been attributed to either impaired copper metabolism, to deficiency of copper in the pasture land or to an excess of molybdenum in the forage.

Several experiments, but most strikingly those of Wintrobe and Cartwright[2] in swine, seem to prove that copper is essential for hemoglobin formation in mammals. It appears from those experiments that copper is necessary for the intestinal absorption of iron, that copper aids in the mobilization of iron from the tissue stores and that copper is required for the proper utilization of iron.

It has been estimated that the optimal daily intake of copper

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* Read at the seventh regional meeting of the American College of Physicians, Santurce, Puerto Rico, October 26, 1957.

** From Fundación de Investigaciones Clínicas and Hospital Mimiya, Santurce, Puerto Rico.

is 1.0 to 2.5 mg. or roughly one tenth that of iron. It has been shown, by the use of radioactive copper (Cu^{64}), that the element is absorbed from the upper gastrointestinal tract, that it is first attached to serum albumin and that within a few hours it shifts to and becomes firmly bound to the alpha-globulin fractions. This copper globulin complex, ceruloplasmin, acts as an oxidase and is involved in the normal process of pigmentation, keratinization, integrity of the nervous system, osteogenesis and in hemopoiesis.

Whole blood and plasma copper are increased in pregnancy, subacute and chronic infections, Hodgkin's disease, acute leukemia, aplastic anemia, hyperthyroidism and hemochromatosis. It might also be increased in chronic leukemia, lymphosarcoma, pernicious anemia, iron deficiency anemias, hypothyroidism and collagen diseases.

Hypocupremia is seen only in the newborn, in nephrosis, in hepatolenticular degeneration (Wilson's disease), in acute leukemia treated with ACTH and, according to Wintrobe, in some cases of non-tropical sprue.

In whole blood studies on 40 normal male subjects, Wintrobe[3] found copper values of 96 ± 13 micrograms per cent and slightly higher values, 100 ± 11 micrograms in 23 normal women. The normal values ranged between 50 and 250 micrograms per cent.

Copper in Soil and Pasture

A number of small but high-grade copper deposits are known to exist on the island. Studies on the amount of copper in the soil were carried out in 1948 by Dr. Juan Ammedee Bonnet, of the Agricultural Experiment Station* in specimens from Canóvanas, Dorado, Caguas and Humacao. The concentration of copper varied from 38 p.p.m. (76 pounds per acre) in Toa Sandy loam of Caguas, to 114 p.p.m. (228 pounds per acre) in the Cayagua Sandy loam of Humacao. Dr. Bonnet also studied some of the common or natural forage on the island, including "malojillo", "Merker", "pasto amargo", "carpet grass", "escobilla", etc. The lowest value, 27 p.p.m. was obtained in "Merker" and "pasto amargo" grasses from Caguas and Canóvanas respectively, while the highest value (57 p.p.m.) was obtained also in "pasto amargo" grass from Canóvanas. These results compare favorably with those reported from continental U.S.A.**

Copper in Fresh Waters of Puerto Rico

The fact that Schistosomiasis Mansoni is so frequent on the island would seem to indicate that copper concentrations should

* Personal communication.

** In U.S.A. soil 5-15 p.p.m.; plants 0.5 to 75 p.p.m.; plants 0.5 to 75 p.p.m.

be low in Puerto Rican fresh waters. Copper is a well known molluscicide. The studies of Harry, Cumbie and Martínez de Jesús[1] of 56 natural waters showed a range of 0.000 to 0.330 p.p.m. copper. The snail Australorbis Glabratus was not found in concentrations of copper that ranged from 0.2 to 0.33 p.p.m. The highest copper concentrations occurred in waters of low dissolved solids. These were the rivers Cibuco and Guayanés (Yabucoa), Marshy pool, the Cidra reservoir and the stream near Juncos.

Material

During the last 2 years we have studied blood copper in 217 healthy Puerto Rican subjects. Determinations on whole blood were made in 147 and in 70 persons the determinations were made on the blood serum. Whole blood copper studies have been performed also in a number of pathological states, including Hodgkin's disease, leukemia, iron deficiency anemias, pernicious anemia, sprue, peptic ulcer and myxedema.

Method

We have used a spectrophotometric adaptation based on the methods and modifications of Warburg (1927), Callan and Henderson (1929), Locke, Main and Rosbash (1932), McFarlane (1932), Tompsett (1934), Sachs, Levine-Andersen and Schmit (1941) and Cartwright, Jones and Wintrobe (1945).[5,6]

Whole Blood Copper

Our series of 147 apparently healthy persons consisted of 100 men and 47 women. In the group of men, there were 19 cases between the ages of 18 and 29 years; 24 in the age group of 30 to 39 years; 25 in the group of 40 to 49 years; 10 in the group of 50 to 59 years; and 22 men were over 60 years old.

Plate I shows the maximum, the minimum and the average values of whole blood copper obtained in each age group. It will be seen that the highest average, 95.3 micrograms per cent, was obtained in the age group of 18 to 29 years; 82.5 micrograms per cent in the group of 30 to 39 years; 75 micrograms in the group of 40 to 49 years; 86.4 micrograms in the group of 50 to 59 years, and the older subjects, those above 60 years of age, gave an average blood copper of 80.9 micrograms per cent. The general average for the entire group of 100 men was 84 micrograms per cent.

The maximum value obtained in the group of men was 290 micrograms and the minimum was 30 micrograms per cent. There were 18 men with whole blood copper values below 50 micrograms per cent.

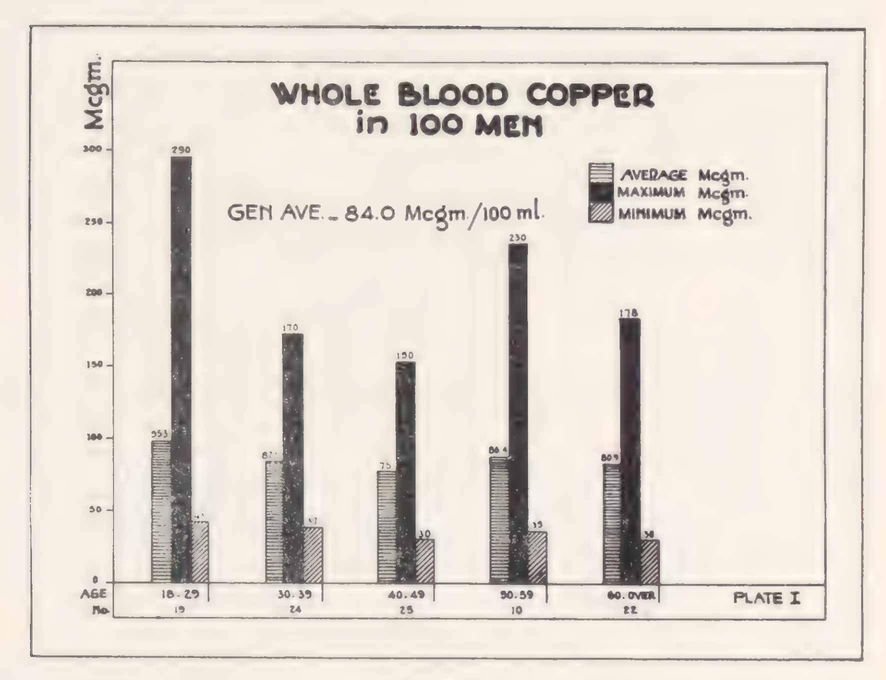


PLATE 1

The group of 47 healthy women, (Plate II), was made up of 9 cases in the age group of 18 to 29, 9 cases in the group of 30 to 39, 10 cases in the group of 40 to 49 years, 6 cases in the group of 50 to 59, and 13 cases were in the age group of 60 and above. The average copper value for the first or youngest group was 94.3 micrograms per cent, 96.1 micrograms for the second group, between 30 and 39 years; 71.2 micrograms in the group of 40 to 49 years; 112 micrograms in those between 50 and 59; and 75.5 micrograms per cent in those of 60 and over. The highest value observed in women was 200 micrograms per cent, the lowest 35 micrograms and the general average was 89.8 micrograms per cent. There were 7 women with copper values below 50 micrograms per cent.

When both men and women were grouped together (Plate III), the general average for whole blood copper in the series of 147 healthy persons was found to be 86.9 micrograms per cent.

Serum copper

Serum copper determinations were performed on 24 men and 46 women: a total of 70 persons. The average in the 24 men of ages of 40 and above was 76.6 micrograms per cent (Plate IV), while that of the women of ages of 18 and above was 83.6 micrograms per cent (Plate V). In the group of men there were 4 sub-

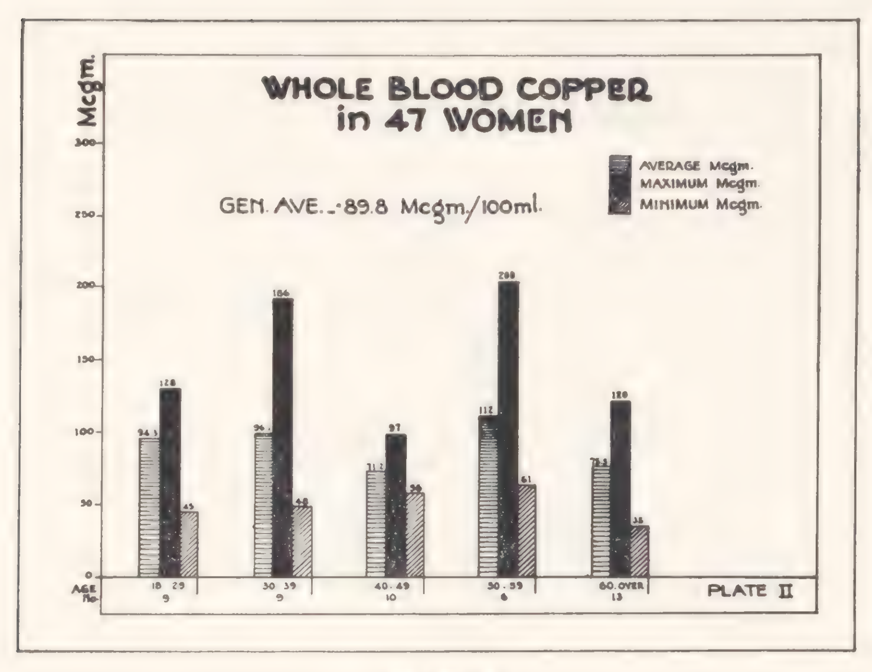


PLATE 2

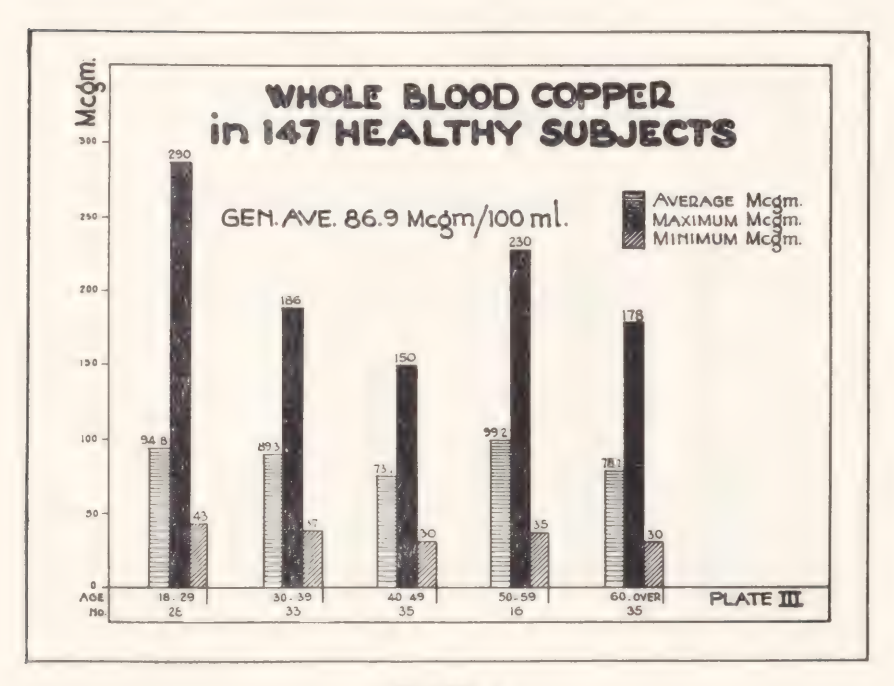


PLATE 3

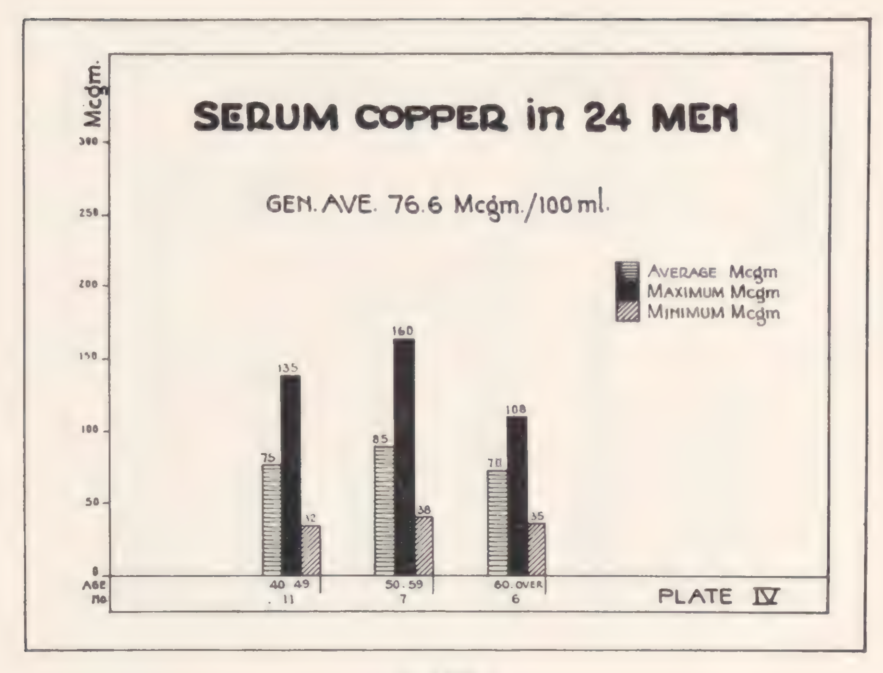


PLATE 4

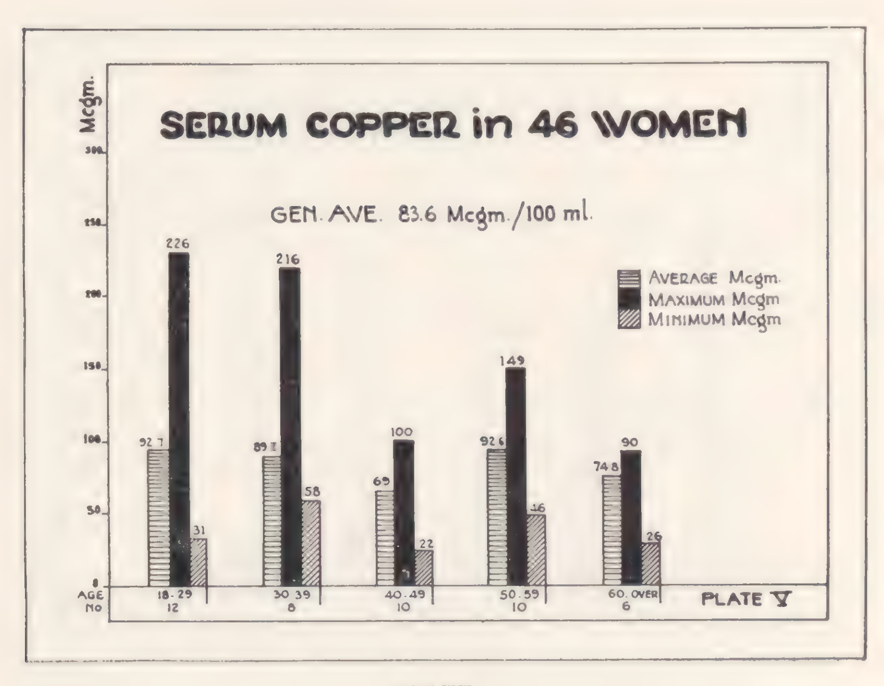


PLATE 5

jects with serum copper levels below 50 micrograms per cent and in the group of women there were 6. The general average for the 2 sexes was 83.0 micrograms per cent.

Lowest copper values

In the entire group of 217 healthy subjects there were 35 (16%) who showed copper values below 50 micrograms per cent. It might be interesting also that the lowest copper values obtained in all groups, except in the small group of 24 men, was that observed in the age group between 40 and 49 years.

Serum Copper in animals.

There is a rather common disease of cattle and pigs observed in Puerto Rico and manifested by paralysis of the hind legs. This disease is known locally by the common names of "derrengue", "ranilla", or "singa". Its symptoms and signs are similar to those of "swayback" and of "falling disease" of Australia and England, but according to Drs. Jaime Bagué and Luis Rivera Brenes, whom we consulted, the etiology of the Puerto Rican disease has not been determined as yet.

With the cooperation of the Animal Disease Eradication Division of the U.S. Department of Agriculture and through the courtesy of Dr. E. R. Mackery, who supplied the blood specimen, we were able to do serum copper determinations on 50 cows, 47 pigs and 19 sheep and goats.

SERUM COPPER in ANIMALS

TABLE I

ANIMALS	NUMBER	Max. Mcgm/100	Min. Mcgm./100	AVE. Mcgm/100
CATTLE	50	310	24	54
SWINE	47	320	45	164
SHEEPS & GOATS	19	256	56	134

TABLE 1

Table I shows the levels of serum copper obtained in the various animals. The blood serum of the cows showed copper levels ranging from 24 to 310 micrograms per cent with an average of 54 micrograms. In the pigs the copper concentration varied between 45 and 320 micrograms per cent with an average of 164 micrograms and in the sheep and goats the range varied between 56 and 256 micrograms with an average of 134 micrograms per cent.

According to Underwood,[7] and we quote: "Normal level of concentrations of copper in the blood of healthy animals is wide, but very similar in all the higher mammals. The normal range for men, pigs, rats, dogs, sheep, cattle and whales, may be set at about 50 to 180 micrograms per 100 milliliters, but a high proportion of values lies between 80 to 120 micrograms per 100 milliliters; with an over all mean not very far from 100 micrograms per 100 milliliters."

Our studies, therefore, reveal normal level for serum copper in swine and sheep, but a definite deficit of the element in the blood of cattle.

Pathological states

Table II shows a few of the pathological conditions in which whole blood copper determinations were made. It will be seen that the highest values were obtained in Hodgkin's disease, 391 micrograms per cent, followed by leukemia, 323 micrograms per cent. In hypochromic anemia the mean value was 208 micrograms per

WHOLE BLOOD COPPER in PATHOLOGICAL STATES

TABLE 2

DISEASES	NUMBER	Max. Mcgm/100	Min. Mcgm/100	AVE. Mcgm/100
HODGKINS	2	488	294	391
LEUKEMIA	2	382	264	323
HYPOCHROMIC ANEMIA	2	216	200	208
PERNICIOUS ANEMIA	1	–	–	176
SPRUE REMISSION	10	272	66	173
SPRUE RELAPSE	8	108	24	60.5
PEPTIC ULCER	12	256	45	94
MYXEDEMA	8	69	37	54

TABLE 2

cent. In one case of Addisonian pernicious anemia we found a whole blood copper concentration of 176 micrograms per cent. The average in 10 patients with sprue in remission, was 173 micrograms, but 8 patients with sprue in relapse showed a low average of only 60.5 micrograms per cent. The average in 12 cases suffering from peptic ulcer was 94 micrograms and the most marked hypocupremia was observed in 8 cases of myxedema with a mean level of 54 micrograms per cent, a minimum of 37 and a maximum of 69 micrograms per cent.

Summary and conclusions

These studies show that although there are small but high-grade copper deposits on the island, and although there is apparently no lack of this element in the soil, in some of the pasture grasses and in a few of the fresh waters of Puerto Rico, the blood level of copper in apparently healthy Puerto Rican people was found to be lower than that reported for continental U.S.A.

In a series of 217 healthy subjects studied, there were 35 (16 per cent) with blood copper values below 50 micrograms per cent.

We found, as others have found elsewhere, that whole blood copper levels are higher than serum or plasma levels, and that women showed higher blood copper values than men.

We also found that the age group between 40 and 49 years showed lower blood copper values than other adult age-groups.

Whole blood copper values were found to be 84.0 mcgm. per cent for men and 89.8 mcgm. per cent for women; while serum copper levels were 76.6 mcgm. per cent for men and 83.6 mcgm. for women.

Copper values were found to be lower in cattle than in pigs, sheep and goats. The mean levels were 54 mcgm. per cent for cattle, 164 mcgm. per cent for pigs and 134 mcgm. per cent for sheep and goats.

In pathological states we found hypercupremia in Hodgkin's disease and in chronic leukemia. In hypochromic anemias normal values for copper were obtained, as well as in pernicious anemia, in sprue in remission, and in patients suffering from peptic ulcers, but rather low levels in patients with sprue in relapse.

Although in the medical literature hypothyroidism is included among the conditions in which hypercupremia might occur, all our 8 cases of myxedema showed definite hypocupremia. No case of nephrosis, nor of hepatolenticular degeneration (Wilson's disease) was studied.

We cannot account for the high incidence of hypocupremia among healthy Puerto Ricans, except on a nutritional base. The

beef from local slaughter-houses, as well as some of the potable water might be low in copper, and the richest sources of copper such as nuts, dried legumes, dried fruits and leafy vegetables such as cauliflower and celery, are rarely, if ever, consumed by the native Puerto Rican.

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THE IMPORTANCE OF FUNGUS SPORES AS AIRBORNE ALLERGENS IN PUERTO RICO: PARTIAL REPORT OF THE FIRST POLLEN AND FUNGUS SURVEY OF THE SAN JUAN AREA

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and
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INTRODUCTION

The study of the airborne allergens of a given region is an important prerequisite toward the intelligent management of the allergic diseases which are found thereon. In spite of the frequent occurrence of these illnesses in Puerto Rico, the investigation of our regional allergens has received little attention. There are only three publications dealing with this subject. In 1941 Quintero Fossas[1] reported on his experiences in the field of allergy in Cuba and postulated that on the basis of similarities in the climatic conditions and botanical flora of both islands, there should be pollens, fungus spores and bacteria in the air of Puerto Rico capable of causing allergic symptoms. In 1946 Toro[2] performed pilot studies on the aerobiology of Puerto Rico and reported the results of occasional fungus cultures done in different localities in the island, including the homes of asthmatic individuals. In 1948[3] Marchand[3] analyzed the botanical flora of the island and listed the potential hay fever plants. We have undertaken a study of the airborne allergens of the San Juan area, using the facilities of the General Medical Research Laboratory of the San Patricio Veterans Administration Hospital. This report will present the results of the first nine months of this study, February through October, 1956.

MATERIAL AND METHODS

1. The standards proposed by the American Academy of Allergy for airborne allergen surveys in the United States have been followed throughout this study. Vaseline-coated glass slides were exposed to the air daily for 24-hour periods in a Durham exposure instrument which was placed on the roof of a one story building in the San Patricio Hospital area. This location overlooks the entire San Juan metropolitan area and can be considered representative of the suburban areas. The findings in this location could be assumed to be similar in the populous north coastal plains of Puerto Rico. Pollen grains and fungus spores falling on these slides were counted over an area of 4.84 sq. cm. and the counts reduced to a standard measuring area of 1 sq. cm. Petri Plates containing Modified Mehrlich's medium for fungus culture were simultaneously

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exposed once weekly for 12 minute periods in three different locations in San Juan, namely: a third-floor balcony facing the Atlantic Ocean in the Condado section, a third-story roof on Ponce de León Avenue and Stop 22 in Santurce, and the same location in San Patricio Hospital where the slides were exposed. The plates were kept at room temperature for 5 days and counts of fungus colonies developing from fungus spores falling on these plates were done. The colonies obtained from the San Patricio Hospital exposures were subcultured on Sabouraud's, Czapeck's, cornmeal, and V8 juice medium, and morphological identification of the genera of fungi was performed.

RESULTS OF THE SURVEY

The 24-hour daily counts of fungus spores far outnumbered those of pollen grains. The fungus counts varied from 2-127 spores per sq. cm., while the pollen counts ranged from 0-12 grains per sq. cm. Although both fungus and pollen counts showed marked daily variations, no seasonal variations occurred in the months studied, February to October, 1956. The predominance of fungus counts, the low pollen counts and the lack of seasonal variations are clearly seen on plotting the monthly totals of these daily counts. (Figure 1.)

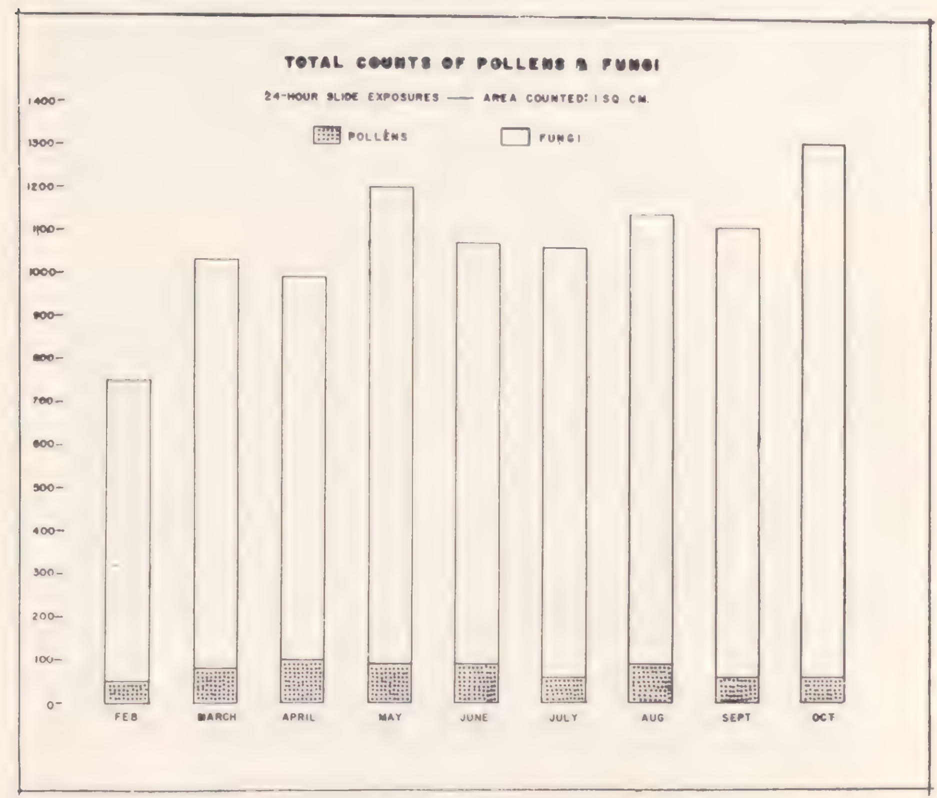


FIG. 1

The simultaneous weekly fungus counts by the Petri plate method showed the following results:

Condado	1-57 colonies
Santurce	3-150 colonies
San Patricio	4-473 colonies

The highest fungus counts almost invariably were obtained in San Patricio and the lowest in the Condado section, the results in Santurce being intermediate. (Figure 2.) The Condado exposure site is representative of the areas of the city near the Atlantic Ocean, the one in Santurce of the "downtown" area, while the San Patricio location represents the suburban metropolitan areas. The results suggest that higher fungus occur in areas where vegetation is more abundant. Similar observations have been reported in the United States by Collier and Ferguson[4] and Wallace, Weaver, and Scherago.[5]

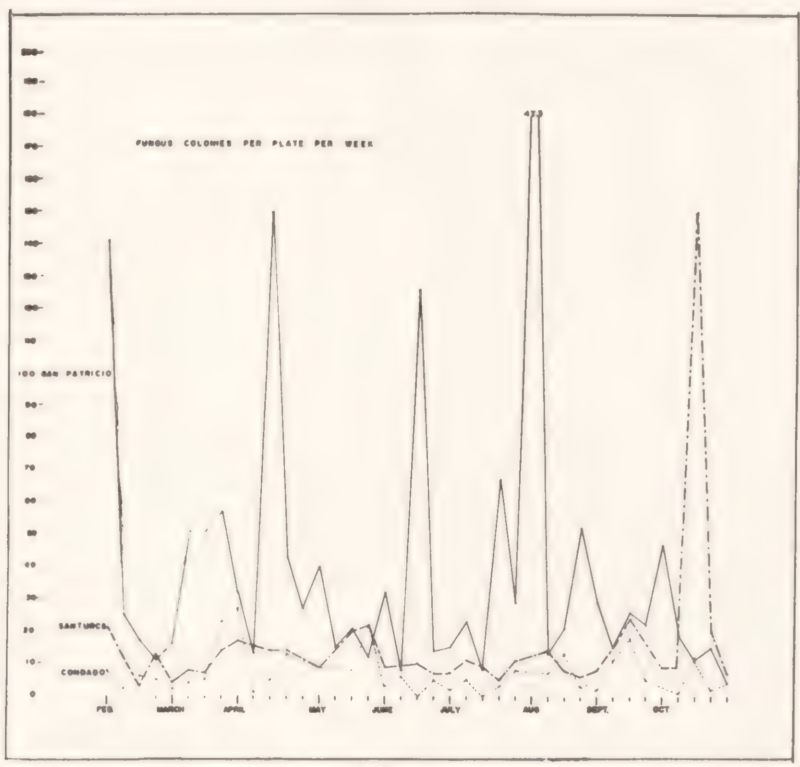


FIG. 2

The most commonly observed genera of fungi were: Hormodendrum, Nigrospora, Candida, Penicillium, Fusarium, Aspergillus, Curvularia, Ustilago, Stemphylium, and Pullularia. Table 1 shows the genera by number of appearance in the plates. These are also the principal ones in respect to total number of colonies.

TABLE I

MOST COMMON GENERA OF FUNGI

1. Hormodendrum	6. Aspergillus
2. Nigrospora	7. Curvularia
3. Candida	8. Ustilago
4. Penicillium	9. Stemphylium
5. Fusarium	10. Yeasts*

* This term includes white and pigmented yeast-like colonies producing no pseudo mycelium.

Botanical classification of pollen grains was not attempted because the low pollen counts did not justify the amount of work implied in this classification. There is no description available of the pollens of our local flora. However, a set of slides exposed in October 1956 was examined by Mr. Oren C. Durham,[6] Chief Botanist of Abbott Laboratories. Grass pollens were the most abundant pollens found on these slides.

The work we have reported has been limited to the San Juan metropolitan area because of the location of our laboratory facilities. We know little of the occurrence of fungus and pollen allergens in other parts of the island. Slides exposed on the same day in San Juan, Aguadilla, and a mountain area near Villalba showed similar pollen and fungus counts.

DISCUSSION

The studies here reported indicate that the spores of fungi are the most frequently occurring airborne allergens of Puerto Rico. Fungus spores are recognized as important causes of allergic diseases in many parts of the world, and they would appear to be the most important airborne allergens of Puerto Rico. Fungus counts done by the plate method in San Juan are higher than those obtained with similar techniques by Feinberg in Chicago, an area where numerous cases of clinical fungus allergy exist.[7]

The pollen counts we have obtained in Puerto Rico are very low in comparison with those from areas where seasonal pollen hay fever occurs. Authorities on the subject[8, 9] state that a total 24-hour pollen count of 6-11 pollen grains per sq. cm. is the minimal number necessary to cause discomfort in an allergic person. In San Juan, the lower limit of this figure was exceeded only in 23 out of 277 days studied. It seems important to us that the upper limit of this minimal figure was reached in only two days of the entire period studied, and was never exceeded. These figures suggest

that in Puerto Rico the pollen count of the air is generally not sufficiently high to produce clinical allergic manifestations. The local maximal daily total pollen count of 12 grains sq. cm. is in sharp contrast with the maximal daily ragweed pollen counts reported by the American Academy of Allergy in various cities of the United States, i.e., Chicago, Illinois 162 grains sq. cm., Pittsburgh, Pa. 347 grains sq. cm., Washington, D. C. 61 grains/sq. cm., Hartford, Conn. 141 grains/sq. cm.[9,10]

Clinical experience with 300 patients with allergic asthma and rhinitis living in Puerto Rico suggests that the majority of allergic patients in Puerto Rico have non-seasonal symptoms.[11] This is in agreement with the lack of seasonal variation in the fungus and pollen counts observed by us. The fact that there are numerous allergic patients in Puerto Rico who complain of exacerbation of symptoms in damp locations and show positive skin test reactions to fungus extracts suggests that these patients may be allergic to fungus spores.

The studies reported have demonstrated the existence of fungus spores as aeroallergens, that is, in the outside air. Considering that local conditions of temperature and humidity are highly favorable for the growth of fungi and that fungi grow easily on numerous places and articles in the interior of home, as mattresses, rugs, drapes, old wood, clothes and shoes, it is entirely possible that individual cases can be exposed to large numbers of fungus spores in the air inside their own homes and places of work. An important aspect of the treatment of these patients should consist of measures designed to reduce the growth of fungus in the home. The use of rugs, drapes, and curtains can be minimized. Plastic or impermeably-sealed covers can be placed on pillows and mattresses. Dense foliage around the house can be thinned to permit sunlight to dry roofs and walls. Closets should be clean and well-ventilated, or a small permanently lit electric light can be installed in them to dry the air. Some of these patients do poorly in old wooden houses. Air-conditioning may help if properly installed to correct excessive humidity and if equipped with adequate filters. The so-called allergy proof foam rubber pillows and mattresses do not harbor dust, but can certainly be excellent sites for the growth of fungi.

Desensitization to extracts made from fungus cultures in an accepted mode of treatment of patients with fungus allergy. Different mold genera vary in their antigenicity, but there is enough cross-immunity among them that the injection of extract from some genera can cause clinical improvement in patients allergic to other genera. Although our work in the indentification of mold genera in Puerto Rico is not complete, the local fungus flora ap-

pears to have certain differences from that of the United States. The genus Alternaria, which is the most frequent, and incidentally the most potent antigen in the United States occupies 13th place in order of number of appearances in Puerto Rico. Certain fungi of frequent appearance here (Nigropora, Curvularia, Candida, and Ustilago) are rare or absent in the United States. We do not know whether any of the locally-occurring fungi have special antigenic importance. For the present, desensitization to polyvalent or stock fungus mixtures seems practical and justified. In some cases, treatment with extracts made from fungus cultures obtained from exposure of plates in the patient's own homes is successful.

Although pollen counts are very low, it must be remembered that there are plants in Puerto Rico potentially capable of producing hay fever. It is possible that sensitive individuals, living in close proximity to such plants, can develop allergic manifestations to them. We believe these cases to be rare.

SUMMARY AND CONCLUSIONS

1. Daily slide counts of pollen grains and fungus spores done in the period February to October 1956 in the San Juan area show that:

a. Fungus spores are much more numerous than pollen grains in the outside air of San Juan.

b. The counts showed marked daily variations, with an absence of seasonal differences, both for fungi and pollens.

c. Pollen counts were generally too low to be of clinical significance.

2. Counts of fungus colonies from plates exposed in the San Juan metropolitan area showed that the lowest values were obtained in locations near the ocean and the highest in suburban areas rich in vegetation.

3. Fungus spores appear to be the most important airborne allergens in Puerto Rico.

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HEMISFERECTOMIA CEREBRAL IZQUIERDA, POR GLIOMA, EN UN NIÑO DE CINCO MESES DE EDAD

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Fué Walter Dandy uno de los más audaces neurocirujanos entre los que iniciaron la universalización de la cirugía neurológica. Amén de otras innovaciones fundamentales a la neurocirugía, fué él quien practicó las primeras hemisferectomías,[6] allá para el año 1928, en pacientes con tumores infiltrantes del encéfalo. Abierto ya el camino a este procedimiento operatorio, siguen su ejemplo otros, hasta que más tarde Krynaw[12] lo hace extensivo al tratamiento de las hemiplejías infantiles acompañadas de disturbios convulsivos. Con el correr de los años la hemisferectomía revela los más diversos factores etiológicos, haciendo del cuadro clínico resultante una indicación perentoria de este recurso quirúrgico; así, los gliomas[1,6,9,10,15,16]; las porencefalias, los quistes intracerebrales y aracnoidales, las microgirias y la esclerosis cerebral,[12] lesiones del parto, meningoencefalitis, el síndrome Sturge-Weber-Dimitri.[3]

Naturalmente, la aceptabilidad de la hemisferectomía —tratamiento harto radical— planteó muchas interrogantes urgentes: ¿Qué del lenguaje si el hemisferio removido es el dominante? ¿Qué de las funciones motoras y sensitivas contralaterales? ¿Cuáles los resultados clínicos con referencia al cuadro original. . . ? El tiempo, la observación y la prudencia selectiva de los casos fueron despejando gradualmente los temores de las primeras intervenciones. Se vió que el niño tenía mayor capacidad vicariante de las funciones cerebrales que el adulto,[4] y que el hemisferio sano tomaba a su cargo las actividades del afectado, convirtiéndose en dominante y subdominante a la vez.[8] Si bien Bell,[1] operando con anestesia local, había tomado la precaución de inyectar novocaína en el área de Broca como medida exploratoria para prevenir una afasia, otros[8 10,14,18] extirparon el hemiencéfalo enfermo indistintamente, fuera el paciente diestro o zurdo, sin repercusiones adversas postoperatorias sobre el lenguaje; terminando Krynaw[12] por decir que el hemisferio removido es siempre el subordinado, porque la dominancia se establece previamente a favor del normal.

Al privar a un ser humano de la mitad de su cerebro, justo era preguntarse cuáles serían los trastornos provocados en la es-

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fera mental. Afortunadamente, pronto se advirtió que la operación no era lesiva para las funciones intelectuales, ni afectaba sensiblemente la estructura de la personalidad preoperatoria.[21,22,23] Por el contrario, era en este aspecto que se obtenían los mejores resultados.[4,18,23] Otro tanto sucedió con los hemipléjicos que padecían estados epilépticos rebeldes a la terapia convencional[8,18,20]. Basándose en su experiencia, Krynaw[12] llegó a la conclusión de que los trastornos mentales y epilépticos, asociados o aisladamente, justifican la intervención hemisferectomizante.

Si bien en los primeros casos de Dandy[6] —todos pacientes adultos con tumores cerebrales malignos— apareció una hemiplejía completa del lado contralateral a la hemisferectomía, no sucedió lo mismo en enfermos jóvenes, sobre todo en niños, y mucho menos en aquellos en los que fué indicada la operación como tratamiento de la epilepsia.[4,5,12] Sin embargo, se ha observado que los residuos paralíticos postoperatorios son más marcados en el brazo que en la pierna[13,17] y más aún en los dedos y en la mano.[17]

Las observaciones referidas a la sensibilidad, tanto superficial como profunda, son ligeramente disímiles. Algunos casos han presentado francas alteraciones en el hemicuerpo opuesto, aunque menos notorias en la cara que en las extremidades; otros han conservado indemnes las sensaciones táctil y dolorosa.[10,12,17] No obstante, los hay que preservan el tacto, el dolor y la temperatura, pero sin poder localizar estos estímulos con precisión,[14] o bien percibiéndolos como una sensación dolorosa retardada que se prolonga por varios segundos;[1] o, más aún, acusando dolor al excitarlos con agentes térmicos sin que puedan distinguirlos como calor o frío.[1] Mientras Mason[11] ha encontrado intactas las sensaciones postural y vibratoria, Bell[1] ha observado una ausencia total del sentido palestésico en los dedos y los ortejos y otro tanto en la discriminación biespacial.

Recientemente Cabieses, Jerí y Landa,[3] del Perú, han descrito una complicación postoperatoria temprana digna de tomarse en consideración. Se trata de un desplazamiento o torsión del tronco encefálico que —provocando alteraciones vasculares, entre otras— puede ser causa de muerte. Agreguemos, finalmente, que entre las secuelas postoperatorias la hemianopsia homónima propablemente es constante.[12,18]

PRESENTACION DEL CASO

Historia Clínica.— El día 26 de enero de 1956 fué hospitalizado en el Departamento de Pediatría (H.M. de S.J.) el niño M.O.C., nacido el 4 de octubre de 1955, de sexo masculino y raza blanca. Cinco días antes había iniciado un cuadro diarreico, pero afebril, con vómitos matinales infrecuentes; esta emesis ocasional no

era del tipo central. La inspección general revelaba una circunferencia craneal mayor que la correspondiente a su edad (44.5 cm.), así como una fontanela anterior abultada. La exploración neurológica fué negativa; sin embargo, el padre declaraba haberle notado en alguna ocasión un estrabismo ocular, lo que no fué comprobado a su ingreso en el hospital ni posteriormente.

Influídos por la frecuencia de hematomas y efusiones subdurales crónicos hallados en lactantes con un cuadro similar al descrito (uno de nosotros, R.C., ha comprobado quirúrgicamente once de éstos en el mismo pabellón pediátrico), se decidió hacer las pruebas de rutina usadas para su diagnóstico, practicando punciones subdurales repetidas y comparando los resultados citoquímicos de las muestras así obtenidas con los del líquido espinal. Esta investigación nos llevó a tres conclusiones: primera, la cantidad de líquido obtenido en el espacio subdural izquierdo era mayor a la obtenida en el lado contrario; segunda, los caracteres físicos del fluído eran diferentes entre un lado y otro; tercera, la composición quimicocitológica arrojaba diferencias notables entre los líquidos subdurales y el espinal.

1.—Líquido subdural izquierdo: células blancas, 20, con 2 polinucleares y el resto linfocitos; proteína cuantitativa, 2,438 mgs./100; azúcar, 44.65 mgs./100.

2.—Líquido subdural derecho: células blancas, 18, con 2 polinucleares y el resto linfocitos; proteína cuantitativa, 234 mgs./100; azúcar, no fué dosificada por falta de cantidad suficiente.

3.—Líquido espinal: células blancas, 2, ambas polinucleadas; proteína cuantitativa, 53 mgs./100; azúcar, 60.7 mgs./100.

El resto de los exámenes —sangre, orina, heces— no tenía valor clínico significativo. Pasados algunos días los trastornos digestivos cedieron desapareciendo las diarreas y los vómitos sin haberse presentado signos de deshidratación. La observación clínica a largo plazo, los estudios puncionales, la hidrocefalia moderada y el abovedamiento fontanelar persistente nos inclinaron a la exploración quirúrgica de la cavidad hemicraneal izquierda, sospechando una efusión subdural o un hematoma crónico.

Acto Quirúrgico.— Día 12 de marzo de 1956. Anestesia endotraqueal con óxido nitroso y pentotal en dosis mínima. Incisión frontoparietotemporal en forma de herradura abierta hacia la oreja. Levantamiento de los colgajos cutáneo y óseo. Corte de la dura siguiendo el borde libre de la abertura craneal, dejándole un pedículo de fijación al mismo nivel de reflexión del colgajo ósteocutáneo. De este modo quedó al descubierto una vasta área del hemisferio cerebral izquierdo, que aparecía constituída en su mayor

extensión por una formación translúcida. Se puncionó ésta, obteniéndose un líquido ligeramente espeso y de color amarillento. Las paredes de la cavidad subyacente aparecían lisas y de color igual al de su contenido. Por delante de esta amplia celda quística se encontraron otras dos, de menor tamaño, que abarcaban poco más de la tercera parte del hemisferio. Hacia la fosa media encontramos el lóbulo temporal adherido a la dura y compuesto de varios nódulos duros y mal delimitados al tacto. El lóbulo frontal estaba ocupado por uno de los nichos quísticos. El lóbulo occipital se hallaba notablemente reducido de tamaño, pareciéndonos incompatible con sus funciones normales. Hacia la línea media se veía una gruesa vena anteroposterior que nacía en la corteza cerebral y desaparecía atravesando la tienda del cerebelo. A la vista estaba la cara mesial del hemisferio derecho.

Analizadas las condiciones expuestas se procedió a la resección en bloque del hemisferio, lo que se hizo a partir de la línea media. Removido el hemicéfalo quedó visible el "muñón central", constituído por el núcleo caudado abrazado al tálamo óptico. Al terminar este tiempo operatorio el niño cayó en un paro respiratorio y quedó sin pulso, accidente que duró unos 20 minutos. Gracias a una laudable labor del anestesiólogo, oxígeno, transfusión de sangre, neosinefrina intramuscular, etc., recobró las funciones vitales.

Curso Postoperatorio.— Veinticuatro horas después de la intervención el niño estaba consciente y alerta, a la vez que tomaba sus alimentos normalmente. En los días sucesivos se notaba una hemiparesia espástica derecha, pero pudimos observar que tenía movimientos voluntarios en ambas extremidades. Presentaba una desviación conjunta de los ojos hacia el lado izquierdo pero ocasionalmente movía ambos globos oculares en sentido contrario. Observamos que al forzar espontáneamente la mirada hacia la izquierda aparecía un nistagmo de cortas incursiones. Las pupilas eran isocóricas y reaccionaban normalmente a la luz. Trece meses después de practicada la hemisferectomía volvimos a examinar el paciente (Fig. 1), anotando los hallazgos siguientes: Perímetro craneal, 51.5 cms. Peso, 10 kilogramos (22 libras). La cabeza tiende a mantenerse en rotación hacia el lado izquierdo. Desviación conjugada de los ojos hacia la izquierda, pero con movimientos libres contralaterales. Paresia facial central derecha. Pupilas isocóricas con reflejos a la luz normales. Pupilas ópticas normales. Paresia espástica derecha, conservando movimientos voluntarios en ambas extremidades, más fáciles en la inferior. Respondía con llanto a los estímulos dolorosos y térmicos, o bien retirando la extremidad. Al colocar el diapasón sobre el maléolo interno derecho respondía quejándose, o bien retiraba la pierna. Signo de Babinski



FIG. 1

y respuesta en abanico bilaterales. (Para esta fecha el niño tenía 18 meses de edad). Signo de Rossolimo intensamente positivo en el pie derecho y moderado en el izquierdo. En cuanto al lenguaje no podemos adelantar opiniones, pues si bien el niño no hablaba cuando se hizo este último examen, me parece necesario un lapso postoperatorio más prolongado por razones de su edad.

Estudio Patológico.— Macroscópicamente la pieza operatoria (Figs. 2 y 3) consistía del hemisferio cerebral izquierdo (8 x 5.5 x 5.5 cms.). Parte de la superficie mostraba circonvoluciones y surcos bien conservados, pero en un área extensa estas estructuras se encontraban borradas y sustituídas por masas blancogrisáceas multinodulares, mal limitadas y de consistencia firme. Al corte se veía una gran cavidad rodeada por quistes satélites menores y con una pared fibrosa bien definida y de color amarillento. El tejido tumoral, de difícil delimitación macroscópica presentaba aspectos variados. En la porción superficial era duro, blanquecino y de aspecto fibroso. Más hacia el centro se tornaba grisáceo, moderada-



FIG. 2

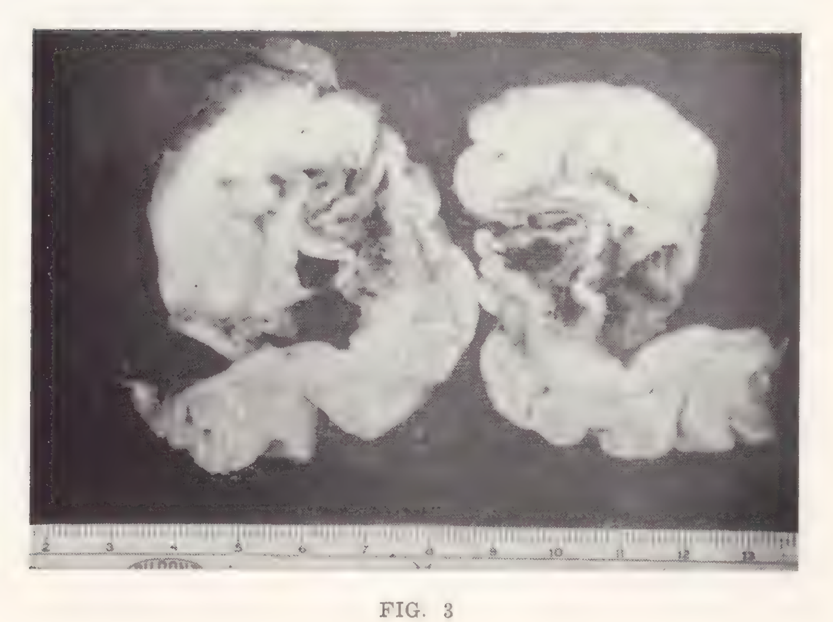


FIG. 3

mente firme y presentaba quistes múltiples de tamaño variable y de contenido gelatinoso o seroso. En la parte profunda se observaban áreas blandas, friables y amarillentas.

Microscópicamente el tumor consistía de una mezcla de tejido neuróglico y conectivo que presentaba tres modalidades morfológicas diferentes con áreas de transición entre ellas: 1—La zona superficial mostraba islotes irregulares de tejido neuróglico bien diferenciado, separados por tejido conectivo fibroso proliferante. En parte de esta zona la proliferación llegaba a predominar sobre la neuróglica en tal grado que recordaba la morfología de un meningioma de tipo fibroso. En la mayor parte de esta zona, sin embargo, tanto los islotes gliales como los tabiques conectivos estaban bien diferenciados y la imagen asemejaba la del llamado "glioma nasal" (Fig. 4). En algunas áreas el componente conectivo del tumor consistía de tejido mixoide más bien que de tejido fibroso. 2—La zona central mostraba quistes múltiples, algunos de ellos conteniendo tejido mixoide, otros vacíos con pared conectiva delgada y mal definida. A medida que disminuía la cantidad de estroma conectivo, las células neuróglicas aparecían más pequeñas e irregulares en tamaño y forma, y la estructura general se asemejaba a la de un astrocitoma grado I de tipo fibrilar (Fig. 5). 3—Unas cuantas áreas, precisamente las zonas friables amarillentas descritas, mostraban tejido tumoral ricamente celular, con elementos fusiformes, y basófilos densamente adosados entre sí de tipo astroblástico. En esta zona se observaban también sistemas glíovasculares así como la formación de empalizadas y de "zonas de devastación" acelulares (Fig. 6). La morfología de estas zonas correspondía a la de un astrocitoma grado II o III.

Tomando en cuenta los hallazgos en la zona anaplásica del tumor, el mismo se clasificó como Astrocitoma grado III.[11] Ahora bien, la variedad de elementos morfológicos observados en el tumor, y su historia, sugirieron la conveniencia de estudios adicionales. Numerosos bloques de tejido fueron cortados y teñidos con coloraciones especiales de anilina y de plata. Se buscó material complementario de otras lesiones patológicas y se enviaron cortes y material a los siguientes consultores: Dr. Isaac Costero (México, D.F.), Dr. J. W. Kernohan (Rochester) y Dr. P. Bailey (Chicago). Los doctores Costero y Kernohan, independientemente, llegaron al mismo diagnóstico de Astrocitoma grado II.[11] Ambos comentaron sobre las peculiaridades morfológicas del tumor, notando la similitud entre algunos zonas de éste y el llamado "glioma nasal".[2] El Dr. Bailey envió la siguiente opinión: "It is a most interesting combination of connective tissue and glial tissue. One could hardly call it a neoplasm; it must be some sort of malformation. I have occasionally seen such a combination in superficial lesions of the

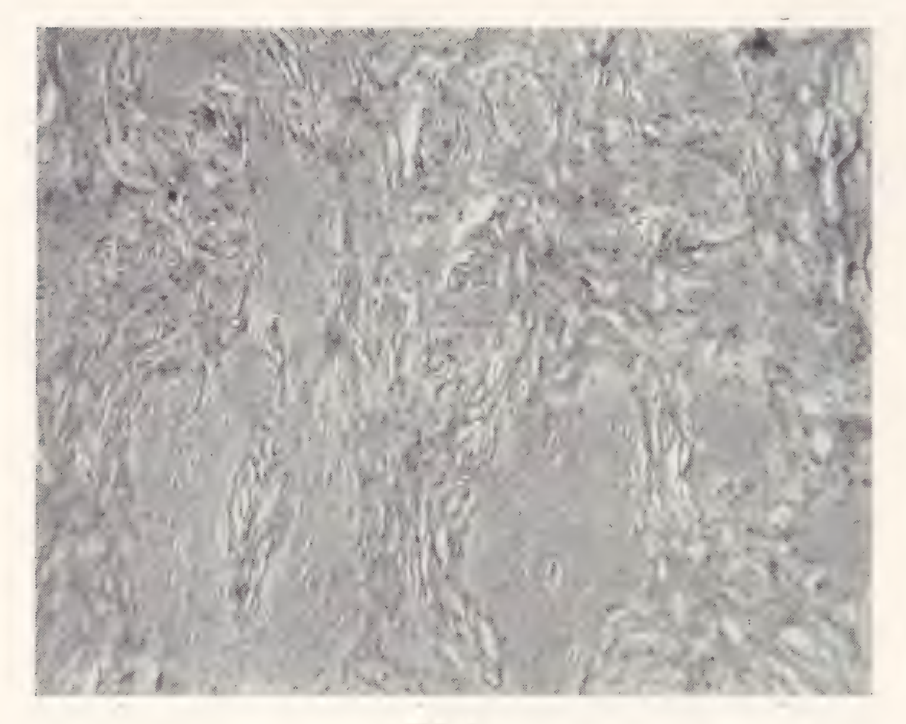


FIG. 4



FIG. 5

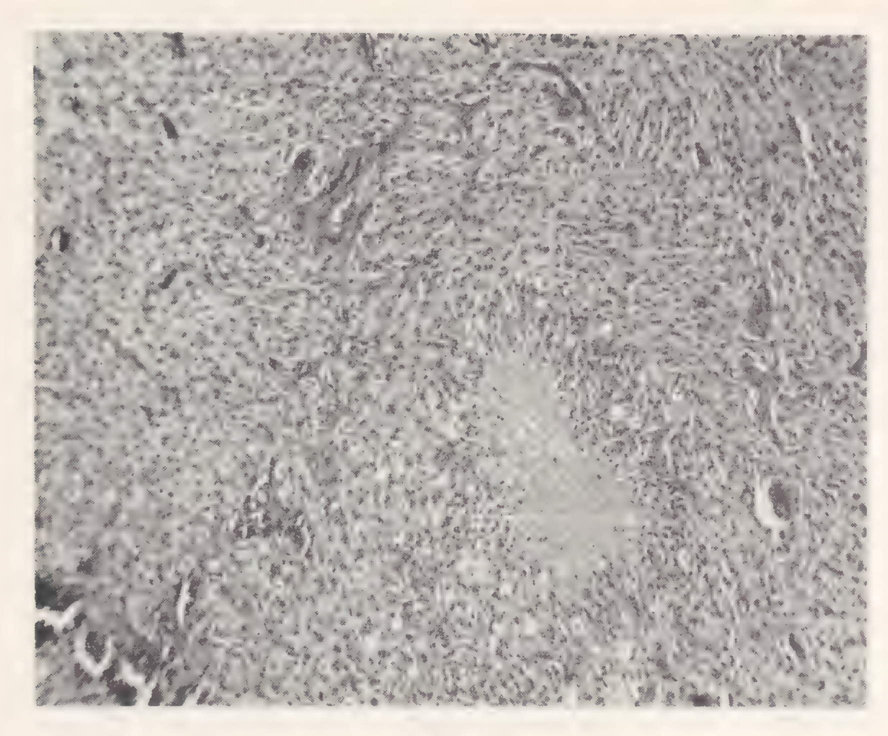


FIG. 6

cerebelium where the neuroepithelial tissue has not differentiated itself very well from the leptomeninx ..."

En lo que se refiere a la técnica operatoria hemos seguido a Obrador Alcalde, quien aconseja la remoción del hemisferio en una sola pieza, partiendo de la línea media hacia afuera;[8,10] contrario a Krynauw,[12] quien lo secciona en cuatro segmentos y completa la hemisferectomía extirpando cada uno de los cuadrantes desde el ventrículo lateral hacia la periferia. Si bien Mensh[16] ha removido el núcleo caudado, el mismo Krynauw[12] que ha tenido vasta experiencia en este procedimiento, cree que esta estructura anatómica, al igual que el tálamo óptico, debe quedar in situ. Este autor ha observado que al extirpar el núcleo caudado la recuperación motora voluntaria del lado opuesto es nula o insignificante; de lo que infiere que es razonable pensar que el hemisferio no removido asume la función motriz ipsilateral por vía del núcleo caudado opuesto. En la literatura consultada únicamente Obrador Alcalde[18] insiste en que el estado general del paciente debe ser vigilado cuidadosamente durante el acto operatorio para evitar el shock, señalando que si la presión arterial desciende de modo alarmante se debe suspender la intervención hasta tanto se restaure aquélla a la normalidad. Como ya hemos anotado en la descripción operatoria, ésta fué nuestra experiencia.

También hemos notado que sólo Cairns y Davidson[1] mencionan la prolongada permanencia —durante meses— del alto nivel proteínico del líquido cerebroespinal. Pudimos comprobar esta observación en nuestro caso, habiendo encontrado en el líquido obtenido del lado intervenido, por punción transfontanelar, siete meses después de la operación, el siguiente resultado: proteína total, 1,400 mgs./100 y sólo 10 células por campo.

Otro aspecto interesante son los hallazgos electroencéfalográficos postoperatorios. En el hemicráneo hueco no hay una ausencia completa de actividad eléctrica, lo que se explica por propagación de ésta desde el lado ocupado.[5] Por el contrario, los registros electroencefálicos tienden a estabilizarse progresivamente dentro de límites normales.[12,17,18] Feld y sus colaboradores[7] han encontrado que lo más común es la débil amplitud del trazado registrado en el lado intervenido. En nuestro caso hallamos asimetría en la amplitud, (Fig. 7) con menores voltajes en el lado izquierdo y descargas epileptógenas derechas.

Aunque los astrocitomas son neoplasias con tendencias al polimorfismo, el cuadro histológico de este tumor está fuera de lo común y merece un comentario especial, opinión con la que concuerdan nuestros consultores. El patrón morfológico de la parte



FIG. 7

superficial del tumor, caracterizado por islotes de tejido neuróglico bien diferenciado entre tabiques de tejido conectivo proliferante (Fig. 4), es difícil de interpretar como imagen de invasión meníngea de un glioma. Más bien sugiere una alteración en el desarrollo de la corteza cerebral y de la leptomeninge con heterotipia de dichos tejidos. Para verificar esta interpretación buscamos otros ejemplos del mismo patrón histológico y encontramos variantes de él en condiciones tan diversas como el glioma nasal y el tejido nervioso que formaba parte de la pared de un quinto dermoide del ovario. La anomalía de desarrollo de la corteza cerebelosa mencionada por el Dr. Bailey puede ser otra variante del mismo cuadro y creemos que con una búsqueda sistemática se encontrarían otros más.

Formulamos la hipótesis de que la presencia del patrón histológico arriba mencionado en una lesión indica que la misma se ha producido sobre la base de una heterotipia previa de tejido nervioso, ya sea debido a trastornos del desarrollo embriológico o como parte de un tumor teratoide. Provisionalmente, hemos propuesto destacar el cuadro histológico ya discutido, denominándolo "coristoglioma", o "patrón coristogliomatoso", con la implicación de un tumor basado en una heterotipia de tejido nervioso. Este concepto está sujeto a comprobación y será objeto de un estudio anatomopatológico más extenso.

Resumen.— Se presentan la historia y los datos bibliográficos principales de la hemisferectomía, exponiendo detalladamente los hallazgos clínicos, quirúrgicos y patológicos de un niño cuyo hemisferio izquierdo fué extirpado a la edad de cinco meses. En el estudio patológico se encontró un extenso astrocitoma grado III, el cual por su cuadro histológico peculiar fué motivo de consultas, concluyéndose que se trataba de una modalidad de crecimiento gliomatoso especial, provisionalmente denominada "coristoglioma" para indicar el origen de un tumor en una heterotipia de tejido nervioso. El paciente ha sido observado por dieciocho meses desde la intervención. Se presentan los hallazgos electroencéfalográficos y clínicos postoperatorios.

Mención de agradecimiento.— Nuestro más respetuoso reconocimiento a la Dra. M. Robert de Ramírez de Arellano, a cuya gentileza debemos el estudio electroencéfalográfico postoperatorio. Los cortes del tumor teñidos con los métodos de Del Río Hortega fueron preparados en el Laboratorio de Patología del Instituto Nacional de Cardiología, México, D.F., por cortesía del Dr. Isaac Costero. Nuestro sincero agradecimiento para él y para los doctores Kernohan y Bailey, que nos ayudaron con sus opiniones autorizadas.

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CARCINOMA OF THE ESOPHAGUS

ANALYSIS OF ONE HUNDRED CASES

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Carcinoma of the esophagus is a dreadful disease which in spite of all the advances in surgical technique continues to baffle the surgeon because of the high[1] operative mortality and the extremely low long term survival.

Who or what is responsible for this situation?

It is not solely due to the patient who waits too long to consult a physician nor to the physician who procrastinates with the radiologic and endoscopic examinations that give the certainty of the presence of the disease. It is due in a considerable number of cases to the disease itself, which develops with disconcerting remissions or produces only late dysphagia. We have seen cancer of the esophagus develop with the patient in good general condition, there existing an extensive neoplasm growing silently. In other occasions the lesions are small but the patients have senile degenerations of the cardiovascular or renal systems or what is even worse pulmonary conditions such as emphysema and chronic bronchitis which make them poor candidates for esophagectomy. The surgeon thus frequently encounters the unfavorable combination of a good general condition with an advanced lesion or a small lesion with a poor operative risk.

In our experience carcinoma of the esophagus is one of the most common if not the most common malignant neoplasm of the alimentary tract.

In an earlier[2] series we reported the high incidence of involvement of the region of the aortic arch and we were then impressed as well as now with the low economic level of the immense majority of the patients.

We have seen one hundred cases of carcinoma of the esophagus during the period between August 1953 and October 1957. Of these cases there were sixty-six males and thirty-four females, a sex ratio of almost two to one.

The age fluctuated between thirty and eighty years, with the highest incidence in the decades of fifty to fifty-nine and sixty-to sixty-nine.

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TABLE I — AGE INCIDENCE

Age	Cases	Percentage
20-29	0	0
30-39	3	3
40-49	17	17
50-59	24	24
60-69	36	36
70-79	14	14
80-89	6	6
Total	100	100

We have excluded from our series lesions of the cervical esophagus. All of our cases had tumors of the thoracic esophagus diagnosed pathologically as epidermoid carcinoma. We have divided the thoracic esophagus into fourths[2] instead of the usual thirds. The lower fourth has been considered as that portion extending from the cardia to the inferior pulmonary vein. The middle half comprises from the inferior pulmonary vein to the aortic arch. The upper half extends from the arch to the thoracic inlet.

TABLE II — LEVEL OF LESIONS

	Cases	Percentage
Upper Fourth	15	15
Middle Half	54	54
Lower Fourth	31	31
Total	100	100

Here we see again the majority of the tumors localized near the pulmonary hilus and the aortic arch. It is possible that the pressure exterted on the esophagus by the aortic arch and the pulmonary hilus predisposes this area of the esophagus to the development of malignancy.

The operability and resectability rates are shown in Table III. The causes of inoperability were wide extent of the lesions, distant metastases and extremely poor general condition. Advanced age has not been a contraindication. We have resected tumors of the lower fourth in two very old patients, one 80 years and the other 82 years old, and both of them have tolerated the operation well.

TABLE III — OPERABILITY AND RESECTABILITY

	Cases	Percentage
Inoperable	54	54
Operable	46	46
Non Resectable	22	48
Resectable	24	52

There is a definite relationship between localization of the tumor and the probability of resection. The tumors of the lower fourth can usually be removed; the ones of the upper fourth are seldom resectable. In his series of a thousand cases of carcinoma of the esophagus, Resano[2] found seventy five of the upper fourth of which only four were resectable. Attention has been called to the increased malignancy of the higher lesions. In the lower fourth, although the inferior vena cava is almost in contact with the esophagus, it is not invaded by the tumor and the adhesions to the descending aorta usually do not go beyond the adventitia. In the upper fourth there is usually a massive periesophagitis that defeats all attempts at resection.

TABLE IV — RELATION BETWEEN LOCALIZATION AND RESECTABILITY

Localization	Cases	Resections	Percentage
Upper Fourth	5	1	20
Middle Half	26	11	42
Lower Fourth	15	12	80

We have approached fifteen cases through the right hemithorax and thirty one through the left. Four resections have been done through the right and twenty through the left. We have used the right sided approach in the cases in which we had planned to reestablish continuity by plastic[3] tube. In three out of the four resections done through the right side, plastic tubes were used. In one case, where almost the entire thoracic esophagus had to be resected because of the extent of the tumor, a plastic tube that extended from the junction of the thoracic with the cervical esophagus to the cardia was placed. The fourth case resected via a right approach was completed by mobilizing the stomach through a separate abdominal incision and making an esophagogastric anastomosis at the level of the thoracic[4,5] inlet. In five of the twenty resections done through the left hemithorax the esopha-

gogastric anastomosis was performed after the esophagus was brought anterior to the aortic arch.[6]

TABLE V — TYPES OF ANASTOMOSIS AND DEATHS

	Cases	Survived	Died	Mortality %
E. G. above arch	5	2	3	60
E. G. at arch	7	5	2	28.6
E. G. below arch[7]	9	7	2	22.2
Plastic Tube	3	2	1	33.3
Total	24	16	8	33.3

E. G. = Esophagogastrostomy

The relationship between mortality and type of anastomosis is given in the above table. A higher mortality is encountered at the higher level. The more proximal the localization of the tumor in the thoracic esophagus, there is increased frequency of adhesions and the anastomosis becomes technically more difficult. In consequence, there is a summation of difficulties: difficulty with the resection and with the anastomosis, more possibility of hemorrhage, ventilatory problems and prolongation of the operation, all of which make the surgical cure more difficult to obtain.

Of the sixteen cases that have survived the operation nine have lived over one year. A present there are three cases living over two years and one over three years.

TABLE VI — CAUSES OF DEATH

	Cases
Anastomotic leak and empyema	2
Coronary Thrombosis	1
Pulmonary Complications	1
Cardiovascular Accident	1
Renal failure	1
Adrenocortical Failure	1
Blood Transfusion Reaction	1
Total	8

Our figures yield a considerable mortality yet this is not unusual when the problem is carcinoma of the esophagus. We have not selected our cases. We have tried and tried very hard to help every case that was operated upon. Our high resectability is a witness to that.

We do not have the pessimism of the others[1] who have gone as far as to advocate resections for carcinoma of the lower third only and to condemn the operations for tumor at other levels.

Our mortality rate could be lowered considerably by eliminating the preventable deaths. The death due to pulmonary complications was the result of accummulation of secretions due to inadequate post-operative care. The one due to renal failure resulted from lower nephron nephrosis due to a transfusion reaction. There was another death due to hemolytic jaundice also the result of a transfusion reaction. If we add to these cases the ones of disruption of the anastomosis, which might have been prevented by a more careful technique in the performance of the anastomosis or by having the patients in better general condition and with higher serum proteins level, we are left with only three deaths; three deaths due to causes beyond the control of the surgeon which would constitute a very acceptable mortality.

SUMMARY

1. An analysis of 100 cases with primary epidermoid carcinoma of the thoracic esophagus has been presented.

2. An operability rate of 46% and a resectability of 52% has been encountered.

3. Twenty four cases have been resected with an operative mortality of 33-1/3%.

4. The causes of death have been analyzed.

5. One of our cases has survived more than 3 years and 3 others more than 2 years after surgical intervention.

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EDITORIAL

NUESTROS OBJETIVOS

El Presidente de nuestra Asociación, Dr. Luis Guzmán López, nos ha encomendado la tarea de dirigir el curso del Boletín de la Asociación Médica de Puerto Rico durante el año 1958. Con la ayuda de la Junta Editora nos proponemos alcanzar ciertos objetivos que forman la base de nuestro plan de reorganización para esta publicación. Creemos que los miembros de esta sociedad deben conocer a plenitud nuestras ideas de manera que nos puedan brindar su cooperación decidida y desinteresada sin la cual estaremos abocados al fracaso.

Nuestros objetivos son los siguientes: 1—Poner el Boletín al día y mantenerlo al día, 2—Levantar el nivel científico del Boletín tratando de aumentar el número de contribuciones médicas y mejorar el calibre de las mismas, 3—Utilizar plenamente al Boletín como portavoz de nuestra Asociación en cuestiones de orden médico, económico, cívico y cultural, y 4—Si es posible, y sin aumentar los gastos de impresión, aumentar la circulación del Boletín entre los centros médicos y las escuelas de medicina de Norte y Sur América.

¿Cómo vamos a lograr estos objetivos? Nuestro primer objetivo de mantener el Boletín al día lo alcanzaremos simplemente si podemos aumentar el número de contribuciones científicas. Este ha sido el escollo inmovible contra el cual se han tropezado todos los esfuerzos de las Juntas Editoras anteriores. Creemos que podremos ganar la batalla mediante el siguiente plan de acción que consiste en abordar el problema de dos maneras diferentes, a saber: 1—Proveer servicio de estenotipista en todas las conferencias médicas de importancia que se celebren en nuestra Asociación de manera que todas las ponencias presentadas puedan ser transcritas rápidamente, sometidas a corrección por el autor y publicadas en el Boletín sin mucha pérdida de tiempo, y 2—Ofrecer gratis un mínimo de 25 reimpresos a los autores de los artículos médicos publicados en el Boletín si éstos así lo solicitan. Es nuestra impresión que la falta de reimpresos ha sido un factor importante en la escasez de artículos médicos, pues muchos autores han creído más conveniente publicar sus resultados en revistas médicas americanas que proveen este servicio.

Hemos presentado nuestro plan ante la Junta de Directores de la Asociación y ésta le ha dado su aprobación incondicional. Pedimos a la matrícula que nos respalde de la misma manera. Si así sucede y con la ayuda del Todopoderoso esperamos alcanzar los objetivos arriba expresados y de esa manera mejorar la calidad de este Boletín.

Agustín M. de Andino Jr., M.D.



Luis R. Guzmán López, M.D.
Presidente, Año 1958

NUESTRO PRESIDENTE

LUIS R. GUZMAN LOPEZ, M. D., *Cirujano Neurológico, nació en San Juan, P. R., el 29 de octubre de 1917, siendo sus padres el señor Manuel Guzmán Carlo y la señora María Luisa López García. Casado con Josefina Vizcarrondo; hijos, Luis Roberto, Josefina, María Amelia y Rosa Margarita.*

Estudios de medicina en la Escuela de Medicina de la Universidad de Maryland, 1936-40. Internado en el Hospital de la Universidad, Escuela de Medicina Tropical, San Juan, P. R., 1940-41. Residencia en el Hospital de la Universidad, Escuela de Medicina Tropical, 1941-43.

Cursos Postgraduados: Fellowship en Patología, Departamento de Patología, Escuela de Medicina Tropical, 1943-44. Curso postgraduado en neurología y cirugía neurológica, Instituto Neurológico de New York. 1945-50.

Patólogo y Director interino, Hospital de Distrito de Fajardo, 1944-45; Profesor auxiliar de neuro-anatomía y neuropatología, Escuela de Medicina de la Universidad de Puerto Rico, 1950; Profesor de Neurología y cirugía neurológica, Escuela de Medicina de la Universidad de Puerto Rico, desde el 1952; Diplomado del Board Americano de Cirugía neurológica, 1952; Cirujano neurológico, Hospital San Patricio, Hospital de la Capital, Doctors Hospital, Hospital de Distrito de Bayamón; Jefe de la División de Cirugía Neurológica de los hospitales de distrito.

Miembro del Congreso de Cirujanos Neurológicos; Asociación Médica Americana, Asociación Médica de Puerto Rico; Fellow del American College of Surgeons; Miembro de la Fraternidad PHI DELTA PI y de la Fraternidad Médica PHI BETA CHI.

En la Asociación Médica ha desempeñado los siguientes cargos: miembro de la Cámara de Delegados, 1950; secretario, 1952; presidente de la Cámara de Delegados, 1956.

De su interés en los problemas médicos en general, su entusiasmo y dinamismo, nos vienen dando fé sus ejecutorias hasta el presente en el desempeño de la presidencia.

Los hombres a cargo del Boletín hacemos votos sinceros por que el más rotundo éxito corone sus esfuerzos en la dirección de nuestra agrupación, y ponemos esta nueva sección a su disposición para que mantenga a la matrícula al tanto de sus gestiones.

SECCION ADMINISTRATIVA

CARTA MENSUAL DEL PRESIDENTE

Salario Mínimo: Deseamos informar a la matrícula que con fecha 18 de febrero entrará en vigor el Proyecto de Decreto Mandatorio Núm. 39, aplicable a la industria de Servicios Comerciales, **Profesionales** y Personales.

Para conocimiento de los compañeros médicos vamos a transcribir la parte del Decreto que se refiere a los Profesionales:

"**Servicios Profesionales:** comprenderá todas las operaciones necesarias o relacionadas con las actividades de servicios profesionales expresamente enumeradas en el apartado (b) de la definición de la industria:

(b) los siguientes establecimientos dedicados a prestar servicios profesionales: bufetes de abogados; consultorios y laboratorios médicos, clínicas y laboratorios dentales..."

"**Artículo III - Salarios Mínimos:** Todo patrono deberá pagar a sus empleados un salario por hora no menor del que a continuación se dispone para las siguientes clasificaciones y subdivisiones de la industria, según se han definido:

Clasificaciones	**Salario Mínimo por Hora**
2. Servicios Profesionales --------	$0.85

De conformidad con las disposiciones de este Decreto los médicos deberán pagar a los empleados que utilicen en sus consultorios un mínimo de 85 centavos la hora.



Asamblea de la Sección Pediatría: La Sección de Pediatría de la Asociación Médica de Puerto Rico, celebrará su Quinta Asamblea Anual del 12 al 15 de febrero. Serán invitados de honor de la Sección en dicha ocasión el doctor Robert B. Lawson, Jefe del Departamento de Pediatría de la Escuela de Medicina de la Universidad de Miami, y el doctor Harry R. Litchfield, Director de Pediatría en el Brooklyn Womens Hospital.

Los actos científicos, que darán comienzo en la noche del MIERCOLES, 12 de febrero, a las 7:30 de la noche, incluirán los siguientes seminarios y conferencias:

Seminarios

- Antibiotic Therapy
- Specific Problems in Infectious Disease
- Steroid Therapy in Children
- Immunization

Conferencias

- Problems Inherent in the Treatment of Children
- Nutrition in the Newborn
- The Evaluation of Vague Complaints

✧ ✧ ✧

Asamblea Anual Asociación de Salud Pública: La Asociación

de Salud Pública de Puerto Rico celebrará su asamblea anual del 5 al 8 de febrero en el Hotel Caribe Hilton.

Durante dicha asamblea se llevará a efecto una discusión a panel sobre el tema "LOS SERVICIOS MEDICOS Y HOSPITALARIOS PARA LA CLASE MEDIA DE PUERTO RICO Y POSIBLES SOLUCIONES A ELLOS", y en el cual participará esta presidencia en representación de la clase médica.

Cordialmente exhortamos a la matrícula para que asista a los actos que se celebrarán en dicha ocasión.



American College of Surgeons - Capítulo de Puerto Rico: El Capítulo de Puerto Rico del American College of Surgeons celebrará su asamblea anual durante los días 26, 27 y 28 de febrero y el 1ro. de marzo.

Trece distinguidos médicos del exterior y 27 de Puerto Rico estarán a cargo del interesante programa científico que se llevará a efecto durante los días de la asamblea. El programa estará en circulación la próxima semana. Espere el suyo.



Medicare: Es con sumo placer que informamos a la matrícula la renegociación del contrato de Medicare. Los doctores Jaime F. Pou y Rafael A. Gil, quienes representaron nuestra Asociación durante las negociaciones en Washington realizaron una gran labor, y próximamente el doctor Pou les informará detalladamente sobre el particular.

El nuevo 'Schedule of Allowances' está siendo confeccionado, y tan pronto se termine será distribuído entre los compañeros que han prestado servicios a MEDICARE durante el primer año de operaciones. Si Ud. interesa rendir servicios en el futuro, favor de avisarlo a nuestra Oficina, de manera que pueda recibir la nueva lista de honorarios.



Código de Seguros: El Comisionado de Seguros de Puerto Rico, señor Pablo J. López Castro, ha obsequiado a nuestra Asociación una copia del Código de Seguros de Puerto Rico, así como el Reglamento promulgado por su oficina para la instrumentación de dicho estatuto.

El precio de dicho Código es de $4.00, y el mismo puede obtenerse en la Oficina del Comisionado de Seguros, Avenida Condado #610, segundo piso. Cuando éste sea ordenado por correo se deberá enviar además 12 centavos en sellos postales. El Regla-

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Escudo Azul: Durante todo el mes de enero hemos seguido realizando gestiones con miras a crear un buen ambiente para los proyectos que permitirán la creación del Escudo Azul en nuestra Isla. El señor Castellucci, Director de los Planes de Escudo Azul en los Estados Unidos continentales, muy generosamente se trasladó a nuestro país y colaboró con verdadero entusiasmo en nuestras gestiones.

Ruego encarecidamente a todos los compañeros que tengan la oportunidad de acercarse a los señores legisladores, que así lo hagan y que discutan con ellos las bondades de nuestros proyectos de ley para establecer el Escudo Azul. Estos proyectos ya han sido radicados y el éxito de los mismos depende del esfuerzo de todos y cada uno de nosotros, demostrando nuestro entusiasmo, nuestro interés y nuestro deseo de que se brinde al público la oportunidad de beneficiarse de este nuevo plan.

A la fecha en que redactamos esta carta hay 385 compañeros que han manifestado estar dispuestos a participar en el programa de Escudo Azul. Sabemos que hay otro gran número de médicos que están dispuestos a colaborar con nosotros en este importante programa, pero quienes por alguna razón se han olvidado de llenar el impreso que le enviamos. Nuevamente acompañamos copia del cuestionario a esta carta con la súplica de que aquellos que aún no lo han hecho, tengan la bondad de llenarlo y enviárnoslo a la mayor brevedad.



Sección de Cirugía General: La Sección de Cirugía General, que preside el doctor Manuel A. Astor, celebrará una asamblea extraordinaria el JUEVES, 6 de febrero, a las OCHO de la noche. En dicha ocasión se procederá a elegir la nueva directiva, se tratarán asuntos de interés general, y se hará entrega de los diplomas a los cirujanos que aún no lo han recibido.



Junta Editora del Boletín: La Junta Editora del Boletín, bajo la presidencia del doctor Agustín M. de Andino, ha trazado un intenso programa con miras a levantar el nivel científico de nuestra publicación, aumentar su circulación y ponerlo al día.

Entre las innovaciones que se propone introducir la nueva Junta Editora, está la de obsequiar a todos los autores con 25 reimpresos de sus artículos.

Cordialmente exhortamos a la matrícula a que envíen sus artículos al Editor en Jefe, doctor Andino.

Nuevos Socios: En la última reunión de la Junta de Directores se aceptaron los siguientes compañeros para formar parte de nuestra agrupación:

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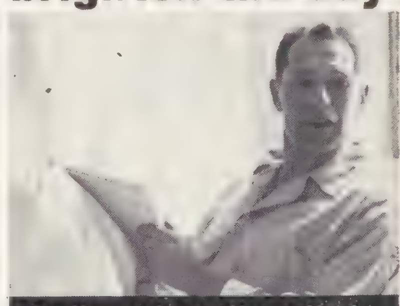
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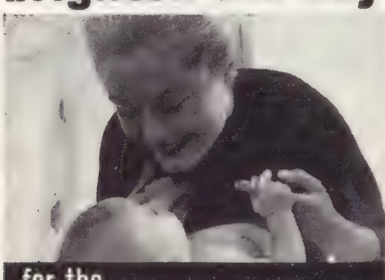
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BOLETIN

DE LA

ASOCIACION MEDICA DE PUERTO RICO

VOL. 50 FEBRERO, 1958 NO. 2

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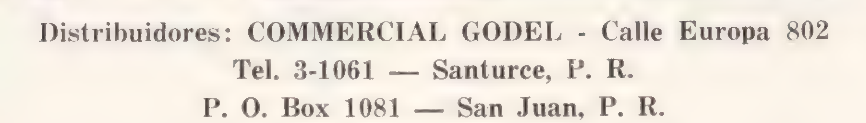
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VOL. 50 FEBRERO, 1958 NO. 2

CLINICAL EVALUATION OF A SIMPLE COLORIMETRIC METHOD FOR TRANSAMINASE DETERMINATION

*MERCEDES VDA. DE TORREGROSA, PH.D.**

Transamination is a chemical reaction governed by enzymes known as transaminases; it consists in the exchange of an amino group from an acid with an alpha keto group from another one resulting in the production of a new amino and alpha keto acids. Many transaminases exist in the human body but the most important ones from the clinical point of view are the glutamic oxalacetic and glutamic pyruvic.

Figure 1 illustrates the chemical reactions involved in the determination of serum glutamic oxalacetic transaminase (SGO-T). The various methods employed are:

1. **Chromatographic**[1] in which the amount of glutamate formed in 3 hours is determined; aspartate and alpha keto glutarate are the amino and keto acids utilized.

SGO-TRANSAMINASE ASSAY METHODS

Chromatographic

aspartate + alpha-keto glutarate $\underset{}{\overset{SGO-T}{\rightleftarrows}}$ glutamate + oxaloacetate

formation of glutamate after 3 hours.

Spectophotometric

aspartate + alpha-keto glutarate $\overset{SGO-T}{\rightleftarrows}$ glutamate + oxaloacetate

oxaloacetic + DPNH + H+ $\underset{dehydrogenase}{\overset{malic}{\rightleftarrows}}$ malate + DPN+

rate of disappearance of $DPNH_2$

Colorimetric

aspartate + alpha-keto glutarate $\overset{SGO-T}{\rightleftarrows}$ glutamate + oxaloacetate

oxaloacetate $\rightarrow$ pyruvate + dinitrophenylhydrazine

color developed

FIGURE 1

* Department of Pathology School of Medicine and Clinical Laboratory, San Juan City Hospital, San Juan, P. R.

2. **Spectrophotometric** method of Karmen et al[2] determines indirectly the transaminase value by measuring the reduction in optical density which occurs when reduced diphosphopiridine nucleotide is oxidized. The oxalacetate produced by the transamination reaction will cause the oxidation of DPNH in the presence of another enzyme, malic dehydrogenase. The optical density of DPN is much less than that of DPNH.
3. **Colorimetric** method[3] measures the color formed when oxalacetate and pyruvate combine with dinitrophenylhydrazine.

The three methods already mentioned: chromatographic,[1] spectrophotometric[1] and colorimetric[5] may be employed in the determination of serum glutamic pyruvic transaminase (SGP-T); the amino acid utilized is 1-alanine instead of aspartate; as a result pyruvate is produced instead of oxalacetate. In the spectrophotometric method lactic is substituted for malic dehydrogenase.

Various modifications of the colorimetric method exist. The colorimetric procedures are less expensive since fewer reagents and the usual equipment found in most routine laboratories are utilized. Recently Reitman and Frankel[6] have devised a very simple colorimetric procedure.

It is the purpose of this communication to evaluate the results of serum transaminase determinations found in various clinical conditions utilizing the colorimetric method of Reitman and Frankel.[6]

MATERIALS AND METHODS

Blood samples were obtained from patients hospitalized in the San Juan City Hospital and from private patients in whom a good follow-up was obtainable. The method employed was that of Reitman and Frankel.[6]*

RESULTS AND DISCUSSION

Normal values reported in the literature for adults (spectrophotometric method) are 8 to 40 units for SGO-T and 5 to 35 units SGP-T. A colorimetric unit of SGO-T is supposed to correspond to 1 spectrophotometric unit[3]; while 1 SGP-T colorimetric unit is equivalent to 1 to 2 units by the spectrophotometric method.[5] In our series among 50 normal persons SGO-T values ranged from 15.6 to 21.6 units and SGP-T from 17.6 to 23 units.

Heart conditions. Among the clinical conditions in which transaminase determinations have been more thoroughly studied

* Reagents obtained from Sigma Chemical Co., 3,500 De Kalb St., St. Louis, Mo.

is myocardial infarction. It has been shown experimentally in dogs[7] that SGO-T levels will rise after coronary thrombosis and that the rise is proportional to the size of the infarct.[8] Infarction of 5-10% of the heart muscle will produce a measurable increase in the serum. Proportionally the amount of transaminase in the infarcted heart muscle is reduced to 2-10% of its original value.

Figure 2 illustrates what occurs in human beings after an attack of coronary thrombosis; serum transaminase begins rising about 6 hours later, reaching a peak within 24 to 48 hours; going down to normal by the 5th to 7th day. If a further rise occurs it is indicative of an extension of the thrombus.[9] It is believed that values above 200 units correlate with a poor prognosis. By taking serial specimens increases in SGO-T in 99% of the patients were described by la Due et al.[10] Values for SGP-T are usually normal; they may be slightly elevated when a large infarct occurs. This correlates well with the finding of 7,100 units pyruvic transaminase per gram of wet tissue in heart muscle in contrast to 156,000 units of oxalacetic transaminase.

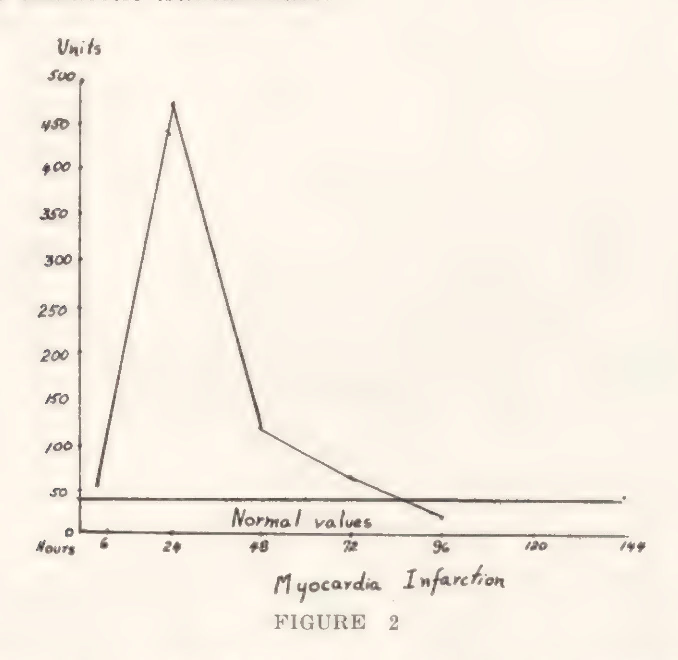


FIGURE 2

Figure 3 depicts the peak values for SGO-T among patients after an acute attack of coronary thrombosis. The first three showed normal values but the tests were done rather late in the course of the illness.

Normal oxalacetic transaminase values were obtained among patients suffering attacks of angina pectoris (Figure 3). This is in agreement with most reports found in the literature. Increases

FIG. 3. MYOCARDIAL INFARCTION

Units	SGO-T	Time
J. C.	17.6	5 days
S. J.	20.4	3 days
J. R.	51.2	4 days
R. P.	51.7	3 days
C. S.	98.0	24 hours
N. P.	104.0	24 hours
J. F.	151.0	24 hours
R. A.	252.0	24 hours
P. C.	484.0	24 hours

ANGINA PECTORIS

11 patients	(18 - 21.6)

PULMONARY INFARCTION

G. D.	18.2

of SGO-T when they occur may represent subclinical findings due to the ischemia.[10]

Most authors report normal values in pulmonary infarction though a large embolus may be associated with a rise in SGO-T,[11] part of the rise may be due to absorption of hemolyzed blood. Prolonged shock may lead to some degree of centrilobar liver necrosis and concommitant rise in transaminase. Multiple emboli may result in a curve similar to that seen after myocardial infarction.[12]

Liver Disease. Studies of the human liver reveal 142,000 units oxalacetic and 44,000 units pyruvic transaminase per gram of wet tissue.[13] Liver disease causes increases in the serum values for these enzymes.

In our cases of cirrhosis (Figure 4) values for SGO-T ranged from normal to 275 units and there were concommitant increases of SGP-T of lesser magnitude. The patients with the higher values died within a short period of time. Other authors report values of less than 300 units for SGO-T in this condition.[13]

Portal hypertension due to Schistosomiasis (Figure 5) was associated with normal, borderline or slightly increased values of SGO-T, SGP-T changes if any were of lesser magnitude. This correlates well with the mild pathological liver changes found in this condition.

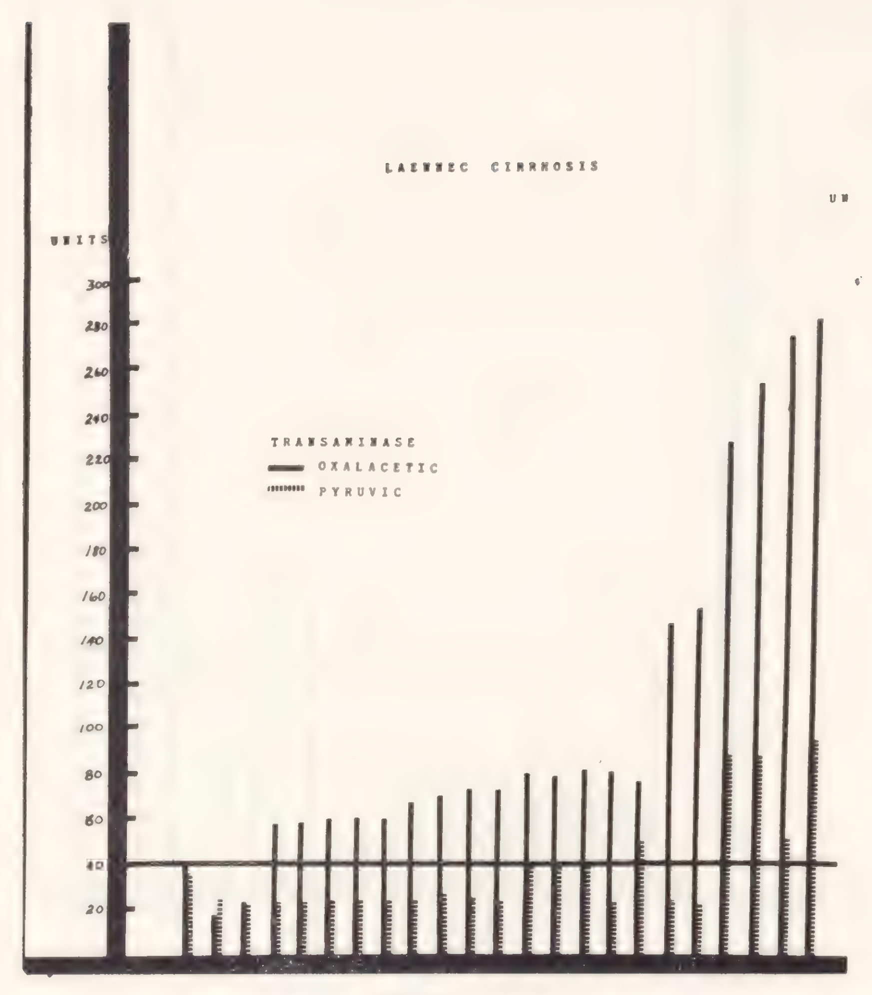


FIGURE 4

Wroblewski and la Due[14] believe SGO-T is a better test than the alkaline phosphatase in the diagnosis of cancer metastatic to the liver; they found elevated values in 23 out of 25 patients. Further experience in this clinical condition has led them to revise this assertion as they have found increasing number of patients with normal values.[15] Rapidly growing malignant tissue is perhaps essential for the production of a high transaminase value. The pattern is similar to the one found in cirrhosis, increases of moderate magnitude for SGO-T and of lesser degree for SGP-T.[13]

Moderate increments in transaminase values are found in extrahepatic obstructive jaundice (Figure 6) with higher figures for pyruvic transaminase in the early stages.[13] Two of our cases had higher SGO-T than SGP-T; at laparotomy they were found to

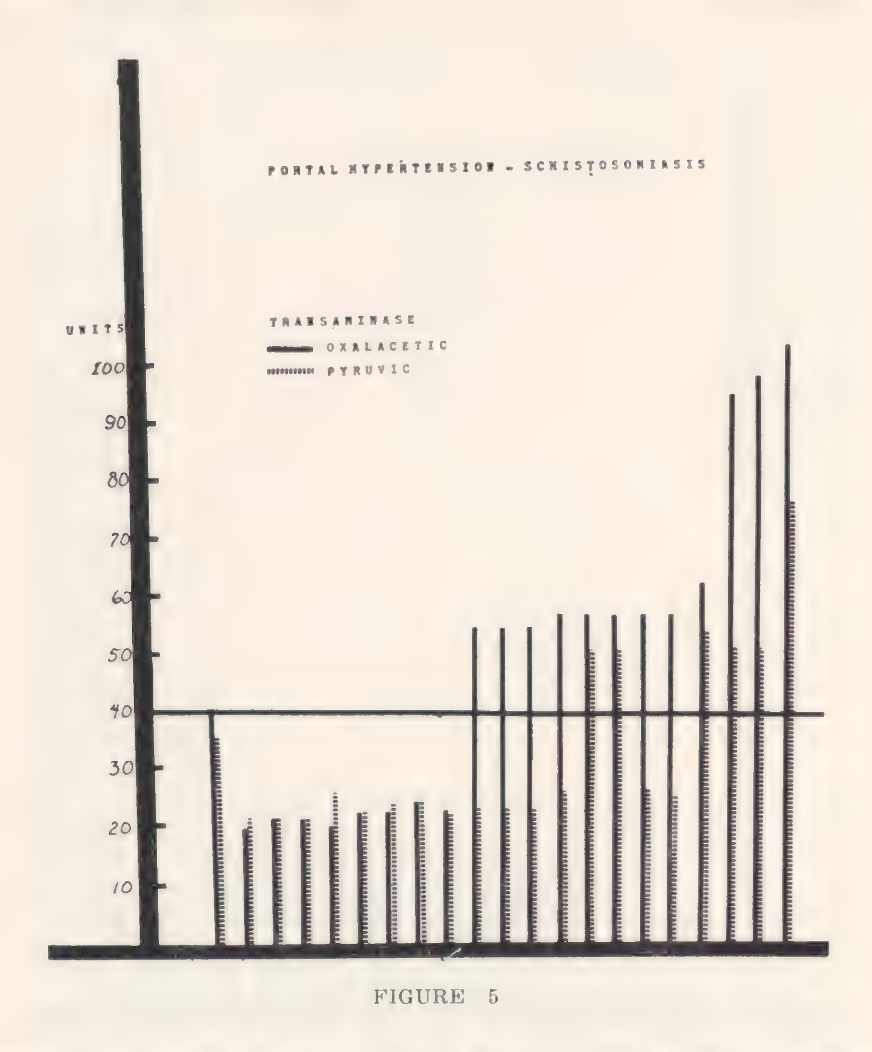


FIGURE 5

have liver metastases. A case of cirrhosis having laboratory evidence suggestive of extrahepatic obstruction is included.

High transaminase values are found in the prodromal stage, as well as in anicteric infectious hepatitis.[16] The values for SGP-T are usually above 500 units and higher than those of SGO-T; changes usually last for 1 to 4 weeks except in protracted cases.[16] Considering that the liver contains 3 times as much oxalacetic as pyruvic transaminase it is difficult to understand the higher SGP-T values found in this disease. Wroblewski and la Due[16] have postulated "variations in release, destruction and excretion of the two enzymes or an unknown metabolic aberration." The newer concept of Shay and associates[17] is also to be considered. These authors are of the opinion that increases of transaminase in liver disease may not be due solely to necrosis of liver cells with re-

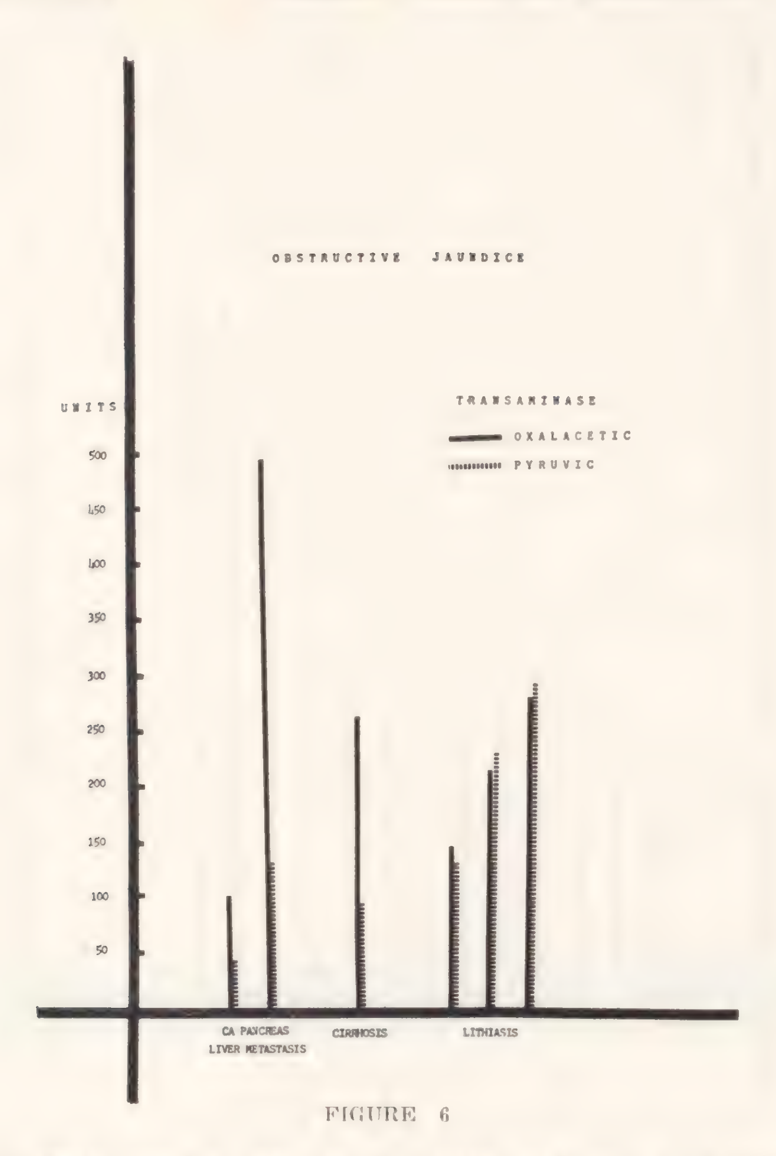


FIGURE 6

lease of the enzyme into the circulation, but also to increased permeability of the liver cells allowing the escape of enzymes. No final answer to the problem is found at the present time.[15]

The values for SGO-T and SGP-T found in our patients diagnosed as suffering from infectious hepatitis are depicted in Figure 7. A case of non-icteric hepatitis is included in which very high values were found (SGO-T 517 and SGP-T 1250 units).

Moderate rises of transaminase are described in infectious mononucleosis with liver involvement.[13] Toxic hepatitis due to thorazine is also associated with a moderate increase in transa-

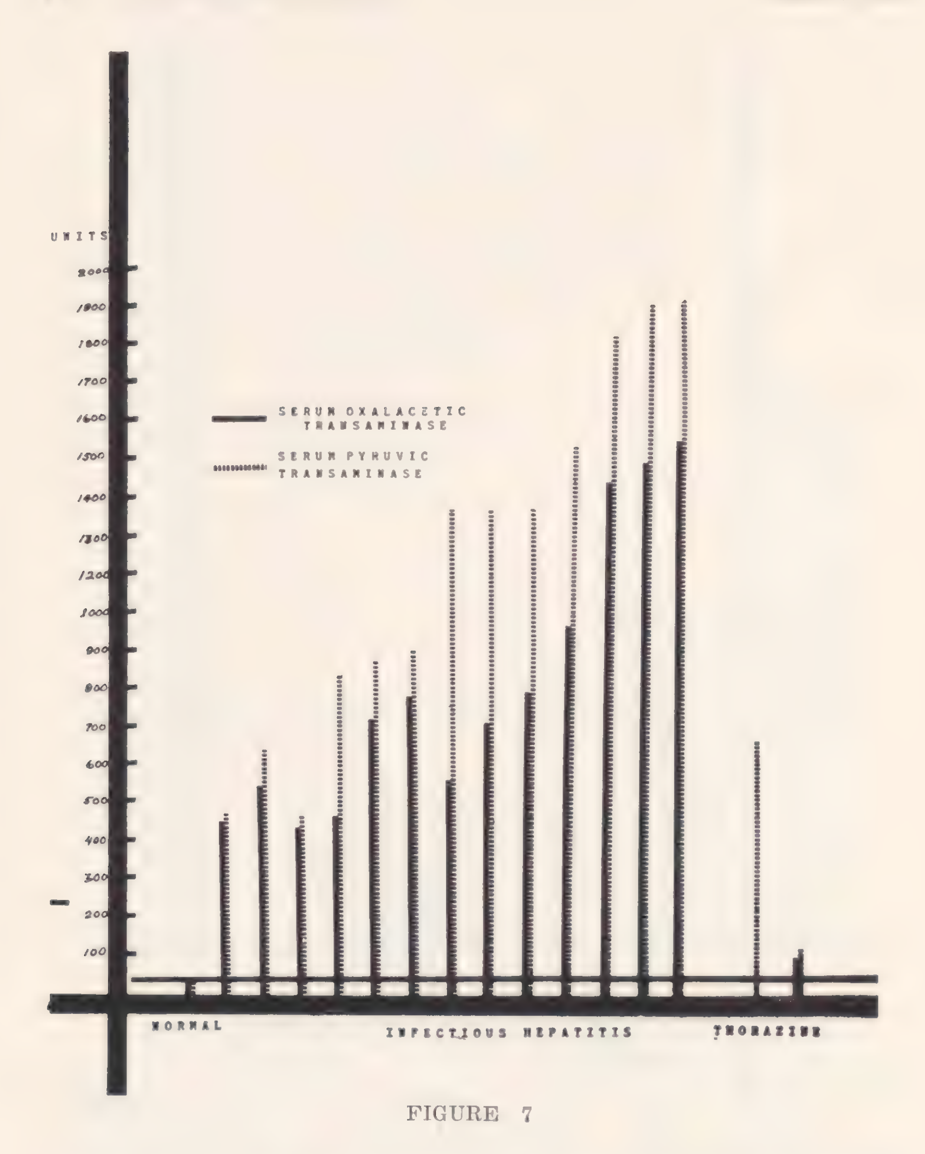


FIGURE 7

minase values. The test is considered a very good index of sensitivity of the liver to the drug.[17]

Figure 8 is a graph correlating the cephalin flocculation, serum bilirubin and transaminase values in a patient suffering from infectious hepatitis who was treated with a prednisolone; the increase in serum transaminases antedated the increment in serum bilirubin at the time of the relapse. To determine the possible effect of meticorten on transaminase values a person with no liver disease, who had been receiving 10 mg. per day for 5 months was studied and normal values for SGO-T and SGP-T were obtained.

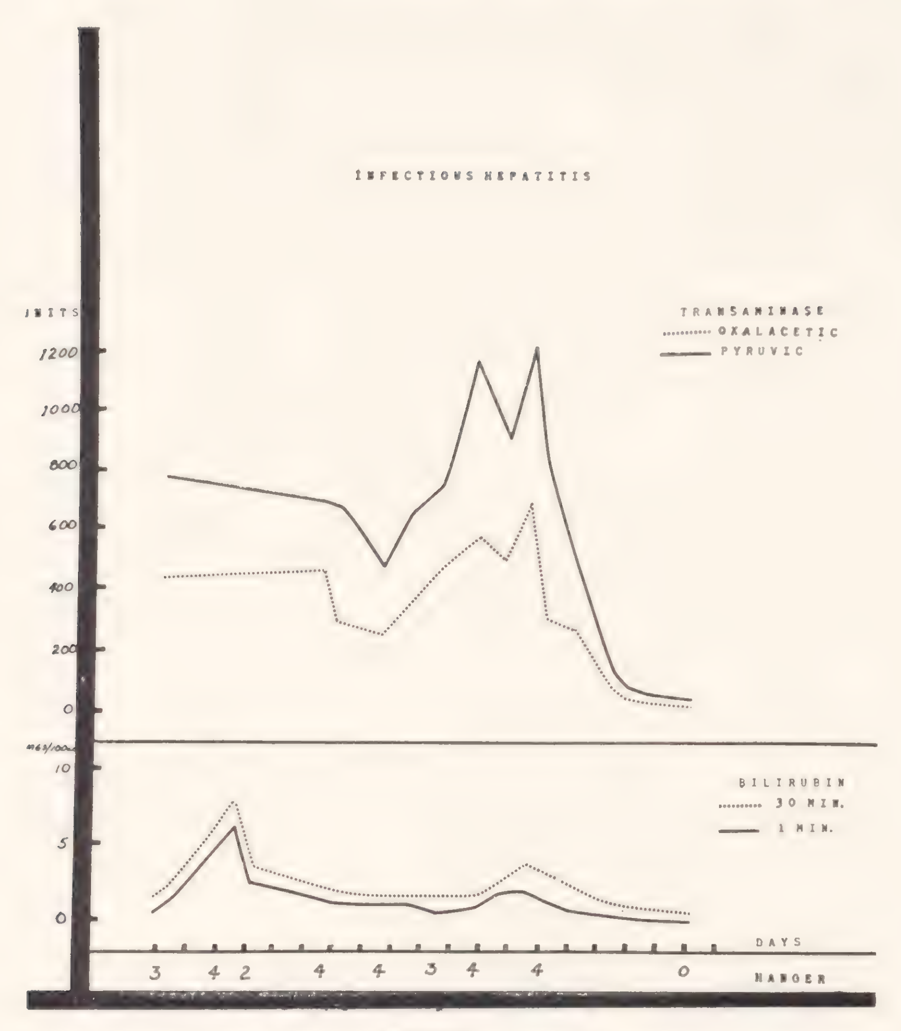


FIGURE 8

Transaminase values by the spectrophotometric method of Karmen et al were compared to those obtained by the colorimetric method of Reitman et al (Figure 9).* Excellent correlation was obtained in the normal and slightly elevated values; among the higher figures some discrepancies were found.

Evaluating the results of SGO-T and SGP-T obtained in our group of patients we believe that the colorimetric method employed gives reliable results that correlate well with the clinical condition of the patients.

* Tests by the spectrophotometric method were performed by Dr. A. Cintrón Rivera of the Clinical Research Laboratory, School of Medicine, San Juan, Puerto Rico.

FIG. 9. SERUM GLUTAMIC OXALACETIC TRANSAMINASE

Spectrophotometric		Colorimetric
16	Units	17
25		21
37		52
53		47
57		54
41		72
114		106
228		151
226		216
288		224
378		252
480		475
272		450
464		673
756		580
516		1019

ACKNOWLEDGEMENT

I wish to express my gratitude to the technical personnel of the Clinical Laboratory of the San Juan City Hospital for their cooperation during the performance of this work.

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HYPERTENSION IN THE PUERTO RICAN AGED

PRELIMINARY REPORT*

RAMON M. SUAREZ SR. and
*RAMON M. SUAREZ JR.***

The Puerto Rican people are also growing old. The average age level is rising steadily, and the proportion of older people is increasing at the same rate. In the year 1940, with a population of 1,877,785, there were on the island only 99,449 people of over 60 years of age (5.3%). In 1945 the number of old people was 117,519, or 5.7% of a total population of 2,048,510. In 1950 the proportion increased to 6.1%: people aged 60 years and over numbered 133,802 and the population of the island was 2,207,028. In 1955 with a total population of 2,263,039 there were 143,722 persons of 60 or more years of age, representing 6.4% of the total. This proportion of old people is still lower than that reported from the U.S.A. To-day there are 14.5 million persons over 65 years of age in U.S.A., which represents 8.5% of the entire population.

POPULATION OF PUERTO RICO

Year	Total	60+Years	%
1940	1,877,785	99,449	5.3
1945	2,048,510	117,519	5.7
1950	2,207,028	133,802	6.1
1955	2,263,039	143,722	6.4

TABLE I

* Read at the annual session of the P. R. Medical Association, Santurce, P. R. Nov. 20, 1957.

** From Fundación de Investigaciones Clínicas and Hospital Mimiya, Santurce, P. R.

During the last few years there has been an increasing interest in the problems of ageing throughout the world. Numerous conferences, commissions, institutes, committees, research projects, etc. have been held. These problems have been only very superficially scratched in Puerto Rico.

We are presenting to-day a preliminary report on the incidence of arterial hypertension in the Puerto Rican aged, based on a 20 years survey of a hospital population. Our studies on the ageing processes of about 300 apparently healthy inmates of an Old People's Home at Puerta de Tierra and our own observations in private patients during the last 30 years will serve as subjects for future presentations.

In the 10-year period from 1937 to 1946 the number of patients admitted to Mimiya Hospital was 7,945. Patients of 60 years of age and over were 549 or 6.9%. Those over 65 years of age were 435 or 5.4% and patients over 80 years of age were only 17, or 0.2%. In the next decade, from the year 1947 to 1956 inclusive, the number of admissions increased to 12,607, but patients of 60 years and over increased to 3,726 or 29.5%. The group of over 65 years increased to 1,583 or 12.5% of the total number of patients admitted and those of 80 and over increased to 164 or 1.3%. It can be seen that although the total number of admissions was not even doubled, there were 6 times as many patients of 60 years

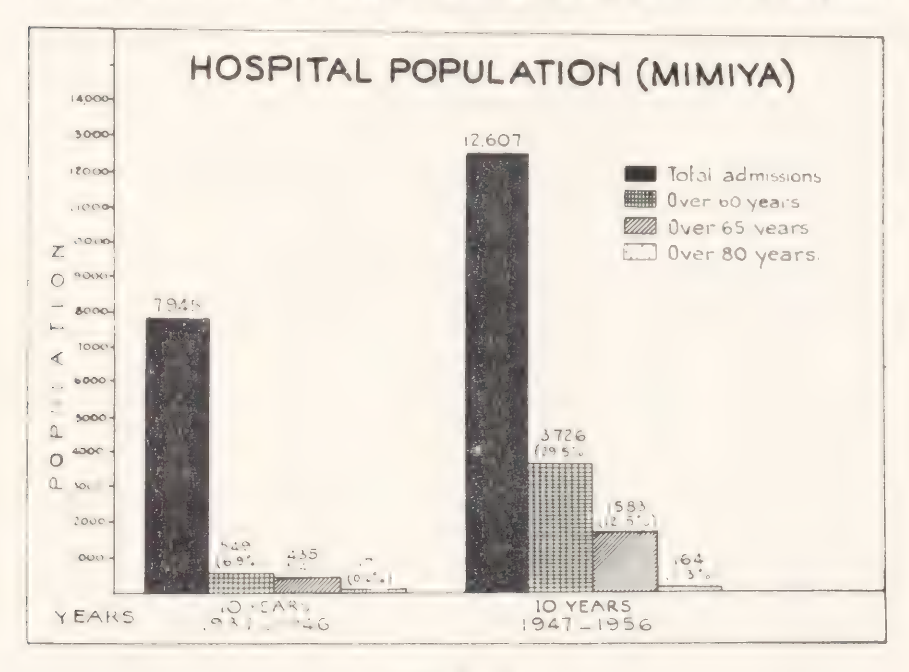


TABLE II

and over admitted in the last 10 year period than in the previous 10 year period.

These figures are more impressive if we consider that a substantial number of patients admitted to Mimiya Hospital are beneficiaries of the Veterans Administration, and that the relatively small number of veterans of the First World War which are supposed to be in their sixties now have been diluted by a large number of middle-aged veterans of the Second World War and by a large number of young men who fought the recent Korean War.

The striking difference in the number of octogenarians admitted; 17 in the first 10 years as contrasted with 164 in the next 10 years, may be accounted for, not only by the increase in life span, but also by the fact of better and more effective medical therapy given to the aged and by better and more successful geriatric surgery.

In the group of 3,726 patients of 60 years and over admitted during the last 10 years, from 1947 to 1956 inclusive, 87% were white and 13% were either negroes or mulattos; 66% were men and 34% were women.

We have reviewed the records of these 3,726 patients and found arterial hypertension in 924 (25.2%). (We considered arterial hypertension any figure above 150 mm. systolic and 90 mm. diastolic). Hypertensive cardiovascular disease with diastolic hypertension of over 120 mm. was found in 118 patients (3.2%) and severe hypertension with sustained diastolic pressure of over 130 mm. was encountered in only 26 patients (0.7%).

In the group of 164 octogenarians (aged from 80 to 96 years), arterial hypertension, most of them benign, was seen in 53 cases (32%), and not a single case of severe diastolic hypertension was observed.

No definite racial or sexual differences were observed in this group of hypertensives, except for the well known fact that women seemed to tolerate the hypertensive state better than men.

Although there were 26 patients who showed diastolic hypertension of over 130 mm. and although in most of them there was a sustained diastolic hypertension of 140 mm., no case of true malignant hypertension was observed among the 924 patients showing high blood pressure.

Discussion — It appears from this study that the incidence of arterial hypertension is lower in the Puerto Rican aged than that reported for continental U.S.A.

Master[1] examined the blood pressure values in 15,000 persons and found that 39% of those between the ages of 40 and 49 years had a blood pressure higher than 140 90. The percentage was found to rise with each decade until the ages of 80 to 89,

PATIENTS 60 YEARS AND OVER		3,726
Arterial Hypertension (above 150/90)	924	(25.2%)
Hypertensive Cardiovascular disease	118	(3.2%)
Severe Hypertension (diastolic over 130 mm.)	26	(0.7%)
Malignant Hypertension	0	
PATIENTS 80 TO 96 YEARS		164
Arterial Hypertension (above 150/90)	53	(32%)
Severe Hypertension (diastolic over 130 mm.)	0	

TABLE III

when 85 per cent had a blood pressure above 140/90. In our group of 164 octogenarians there were only 32% hypertensives, when we used 150/90 mm. Hg. as the upper limit of normal blood pressure values, but even when using his figures of 140/90 mm. Hg., the percentage of hypertensives did not go above 54%.

The great majority of our elderly hypertensives showed only simple benign systolic hypertension. This is the common uneventful form of high blood pressure which Evans[2] from Britain terms "hypertonia". A number of our aged showed the hypertensive cardiovascular disease characterized by retinal artery narrowing and electrocardiographic evidences of left ventricular hypertrophy, and a few of our aged Puerto Ricans showed the severe form of arterial hypertension characterized by a sustained diastolic hypertension of over 130 mm. of Hg. with renal, cardiac or cerebral manifestations. A few of these patients are alive 10 years after the diagnosis was established, while without treatment, death within a year is the rule in malignant hypertension.

As already stated, no case of true malignant hypertension was observed in our group of 924 elderly patients. Sometimes it is difficult to distinguish true malignant hypertension from the severe types of essential hypertension, and from hypertension secondary to known hypertensive diseases, such as chronic glomerulonephritis. True papilledema is, we believe, an essential criterion for the diagnosis of malignant hypertension. In doubtful

cases, a lumbar puncture will help to clear the diagnosis. There will be a cerebrospinal fluid pressure of 300 mm. or more in malignant cases, (except in terminal heart failure), in contrast to about 200 mm. in the benign cases.

Conclusion — The number of patients of 60 years and over admitted to a general hospital in Puerto Rico has gradually and progressively increased during the last 20 years, pari pasu with the rising number of older people in the population of the island.

While in the 10 year period from 1937 to 1946 only 6.9% of the hospital population were people of 60 years and over, in the 10 year period from 1947 to 1956 the proportion of old people admitted to the hospital increased to 29.5%.

Using as upper limit of normal blood pressure 150 90 mm. Hg. we found that 924 of 3,726, or 25.2%, old people admitted to the hospital showed an elevated blood pressure. Real hypertensive cardiovascular disease was observed in 118 patients (3.2%), and severe hypertension with a sustained diastolic pressure of over 130 mm. was seen in only 26 instances (0.7%).

In the oldest group of 80 to 96 years, there were 164 patients. Arterial hypertension was observed in 53 (32%), but there was in it, not a single case of severe hypertension with diastolic pressure of over 130 mm.

In a hospital population of 924 hypertensive patients, no case of malignant hypertension was observed.

✧ ✧ ✧

Conclusión — Ha habido un aumento gradual y progresivo en el número de pacientes de más de 60 años de edad admitidos durante los últimos 20 años a un hospital general en Puerto Rico. Este aumento ha ido pari pasu con el aumento en la proporción de gente vieja en la población de la isla.

Mientras que en el decenio comprendido entre los años 1937 y 1946, el número de enfermos de más de 60 años de edad representó solo un 6.9% del total de admisiones al hospital, esta proporción aumentó a 29.5% en el siguiente período de 10 años (1947-1956).

En ese último período y aceptando como límite superior de la tensión arterial normal 150 90 mm. de mercurio, encontramos hipertensión arterial en 924 del total de 2,726 sujetos de más de 60 años de edad (25.2%). De ese número, solo 118 enfermos (3.2%), presentaron evidencias de enfermedad cardiovascular hipertensiva, y en solo 26 pacientes (0.7%) encontramos una tensión diastólica sostenida de más de 130 mm. de mercurio.

En el grupo de individuos de 80 a 96 años de edad, representado por 164 pacientes, encontramos hipertensión arterial en

53 (32%), pero en ninguno de ellos la tensión diastólica fué de 130 ó más mm. de mercurio.

En la serie de 924 pacientes hipertensos de más de 60 años de edad no apareció ni un solo caso de hipertensión maligna.

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STAB WOUNDS OF THE HEART

REPORT OF SIX CASES

*MARINO BLASINI RIVERA, M.D.**
*FRANCISCO L. RAFFUCCI, M.D.**

When a heart chamber is entered the wound bleeds rapidly into the pericardial sac and the pleural cavity. Rarely do these patients exanguinate, but more often 100-200 cc. of blood collect in the closed pericardial sac giving rise to compression of the heart. As the pressure rises there is interference with the afferent blood flow to the heart. Since less blood returns to the heart from the venae cavae and pulmonary veins and because of the compression of the organ a great diminution in stroke and minute volume occurs. The cardiac output falls precipitously and the work of the cardiac muscle is greatly impeded. At times, rather than obtaining an increase in venous pressure as is seen in chronic constrictive pericarditis, there is no change. The explanation is that in acute tamponade the ventricles as well as the atria are compressed whereas in chronic pericarditis the obstruction is at the atrioventricular groove resulting in good ventricular contractions and poor systemic venous return.

The above are the physiopathologic changes that result in improper oxygenation producing air hunger in the presence of a normal blood volume.

Although stab wounds of the heart were recognized by Homer, no cases had been successfully treated till Rehn (1896) performed the first successful cardiorrhaphy.

In 1954 the first case of stab wound of the heart was recognized ante-mortem in the San Juan City Hospital and treated successfully in our surgical service. Since then a total of 6 cases has been diagnosed and treated by our group. At this juncture we wish to present this short series in the hope that we can raise your interest in this condition.

TABLE 1

SIX CASES OF STAB WOUNDS OF THE HEART

Males	5
Females	1

There were 5 males and one female in this group whose ages ranged from 19 to 62.

* From the Department of Surgery, San Juan City Hospital.

It is interesting to note that there were two attempts of suicide while the rest were attempted homicides. The favorite instrument for the incision was found to be a knife although an ice-pick and a sharp thick wire were used also.

The diagnosis of acute cardiac compression is the most important factor in the treatment of these patients. It is of extreme importance to recognize shock, as manifested by hypotension and imperceptible pulse, in association with a precordial or upper abdominal stab wound and an extreme air hunger which is manifested by cerebral symptoms of irrationality, mania, tachypnea and a desire to sit up. Distended neck veins may be observed as well as cyanosis.

On physical examination the heart sounds are distant, as usually seen in shock. The cardiac dullness is not increased.

Needless to say that the future of the patient depends on the perspicacy of the admitting officer who must suspect every chest injury as a possible heart wound.

In all of our cases the diagnosis of stab wound of the heart with acute cardiac tamponade was made tentatively prior to thoracotomy. It is well to state that roentgen examination is of no help because of the urgency of the problem and because of failure to reveal changes in the size and shape of the heart. It was performed in three of our cases and no additional information was obtained.

TABLE 2
SITE OF THE WOUND

Left ventricle	4
Right ventricle	1
Left atrium	1

The site of injury is depicted in table 2 and we notice that the left ventricle was entered in four instances, and in two patients there were lacerations of the lung and large pulmonary vessels.

The operation was carried out as soon as the diagnosis was established without going through hospital routine such as admissions, hemograms, etc. A thoracotomy on the site of the stab wound was performed through that interspace, but we never hesitated to section the sternum transversely and enter the other hemithorax.

TABLE 3
RESULTS

Operative deaths	1
Recovered	5
Late deaths due to sepsis	2
Survivors	3

Table 3 shows our results. Although one patient did not recover from the wound and died in fibrillation in the operating room, five of them survived two weeks or more. As is the case in this type of injury late mortality due to septicemia is rather high and accounted for two deaths. One was due to cerebral abscess two weeks postoperatively. The other died of empyema and purulent pericarditis six weeks later in an insane asylum.

The three surviving patients are well and asymptomatic at present. Thus our uncorrected mortality compares well with that of most observers, about 50% of the patients.

JUVENILE RHEUMATOID DISEASE

REPORT OF FIVE CASES

*MARGARITA C. COSTAS, M.D.**

Juvenile rheumatoid disease is not a rare condition. The incidence of new cases per year has been compared with that of nephrosis.[1] Five per cent of all the patients with rheumatoid disease belong in the ages below fifteen years, that is, in the juvenile group.

The term Still's disease, previously applied to those patients in whom splenomegaly, generalized lymphadenopathy and anemia are prominent features, has fallen into disrepute. The arthritis is but one component of the systemic disease and for this reason the term rheumatoid arthritis is misleading.

The purpose of this report is to present and discuss five cases of juvenile rheumatoid disease admitted to the Department of Pediatrics of the San Juan City Hospital from November 1953 to November 1956. Through this presentation we expect to illustrate the fact that juvenile rheumatoid disease in its onset is usually accompanied by a violent systemic reaction and that the clinical manifestations are extremely protean. The diagnosis of such a disease is at times a real challenge to the pediatrician.

Patients I and II will be presented in detail since they are more illustrative of the clinical picture seen in the pediatric age group.

Case I: A. L. D., was an 8 year old white male admitted on November 18, 1953. Three days prior to admission he had sudden onset of sore throat and arthralgia involving the knees and ankles. On admission he also complained of pain over the sternal area and right hemithorax on deep inspiration. The present illness was accompanied by high fever. There was no history of previous similar episodes. The physical examination on admission revealed a pulse of 140 beats per minute, a rectal temperature of 103º F, and a blood pressure of 120/80. The tonsils were hypertrophied and hyperemic. There was marked generalized lymph node enlargement. Examination of the heart revealed the point of maximum impulse in the 6th left intercostal space, one centimeter to the left of the midclavicular line. The rhythm was regular and a grade I systolic murmur was heard over the pulmonic area. The lungs were clear to auscultation. Abdominal

* From the Department of Pediatrics of the School of Medicine and the San Juan City Hospital.

examination revealed no hepatomegaly or splenomegaly. The joints showed no signs of inflammation. The laboratory determinations presented the following findings: a complete blood count on admission showed a hemoglobin of 58% of normal, a white blood cell count of 55,800, a differential of 2% eosinophiles, 5% stabs., 88% polymorphonuclear and 5% lymphocytes. The red blood cells were microcytic and hypochromic. During the hospital stay the hemoglobin varied from 36-62% of normal and he had a persistent granulocytic leukocytosis. The examination of the urine showed a specific gravity varying from 1.000 to 1.020, a negative microscopic examination and occasionally traces of sugar and albumen. Two sedimentation rate determinations revealed values of 32 mm/hour on November 20, 1953 and 25 mm/hour on January 14, 1954. Repeated blood cultures were negative except for a positive culture for hemolytic staphylococcus aureus obtained on November 18, 1953. Febrile agglutination tests were found negative. The total serum protein varied from 7.2 to 8.2 grams per cent. The albumen-globulin ratio was inverted. The bone marrow examination was negative for malignant cells, L. E. cells, and malaria organisms, the red blood cells were hypochromic. Hookworm and trichuris ova were seen in the stool examination. The stool culture was negative. A total eosinophile count determination showed 44 eosinophiles per cubic millimeter. A cephalin flocculation of +1 in 24 hours and +2 in 48 hours was obtained on December 15, 1953. The alkaline phosphatase was 5.4 Bodansky units.

A X-ray film of the lungs done on admission revealed a pneumonitis in the right base. Three days later there was resolution of the pneumonic process and the right ventricle was found prominent. A cardiac chamber analysis done on November 27, 1954 presented no evidence of chamber enlargement. X-rays of the wrists and ankles done on November 25, 1953 were interpreted as indicative of soft tissue swelling but there was no evidence of osseous pathology. The forearm and legs showed no evidence of osseous pathology. On March 17, 1954, X-ray films of both hands revealed soft tissue swelling in the region of the wrists with marginal bony destruction of the central carpal bones and proximal ends of the second, third and fourth metacarpal bones.

An electrocardiogram performed on November 20, 1953 was interpreted as indicative of sinus tachycardia with auricular and ventricular rates of 167/min. and changes compatible with myocardial damage. Electrocardiograms done on December 21, 1953 and March 2, 1954 were negative except for sinus tachycardia.

From admission on November 18, 1953 and until December 18, 1953 no definite diagnosis could be established. The following conditions were considered: myocarditis, cause undetermined, septice-

mia due to Micrococcus pyogenes var. aureus, malaria, typhoid fever, brucellosis, and leukemia. During this time he received the following antibiotics in succession: penicillin, erythromycin, terramycin, and streptomycin. He also received one course of treatment with Aralen. On two occasions he developed a generalized urticarial rash that was interpreted as drug allergy and treated with pyribenzamine.

One month after admission he developed redness and swelling of the wrists and ankles and fifteen days later he was found to have fusiform swelling of the right third finger. At that time the liver was palpated 2 cm. below the right costal margin. The diagnosis of rheumatoid disease was then made and because of the severity of the illness the patient was started on cortisone 200 mg. daily. The following day he became afebrile for the first time and remained so until January 14, 1954, one day after the cortisone was discontinued because the patient had developed edema and hypertension. From January 30, 1954 to March 21, 1954 he received ACTH. On March 31, 1954 he was again started on Cortisone because of signs of activity. The dose was gradually tapered off without complications and on July 30, 1954 the patient was discharged and referred to the Crippled Children Clinic. He never had a true remission but became severely crippled and died two years after the onset of his illness. An autopsy was not done and the immediate cause of death is not known.

Case II: A. R. C., a 7 year old white male who had been in a private hospital for 2 weeks and was referred to the San Juan City Hospital on October 9, 1955 with the following history. On August. 1955 he had sudden onset of high fever, profuse sweating, anorexia and generalized muscle and joint pain. A week later he was admitted to the hospital. The physical examination on admission revealed a rectal temperature of 103º F., a pulse of 100 beats per minute and respirations of 34 per minute. The tonsils were enlarged and cryptic. The heart was enlarged and a grade III apical systolic murmur was heard. There was impairment of resonance at the base of both lungs. Examination of the abdomen revealed no visceromegaly and the joints showed no signs of inflammation.

Electrocardiographic studies revealed changes compatible with pericarditis. Throat culture grew B. hemolytic streptococcus. A presumptive diagnosis of rheumatic fever with heart involvement was made for which the patient was treated with aspirin and cortisone prior to admission to our department. He also received penicillin, sulfadiazine and terramycin but did not improve.

Physical examination on the first admission to the San Juan City Hospital revealed a rectal temperature of 103º F., pulse of

120 minute, and blood pressure of 85/50. The tonsils were enlarged and hyperemic. The eyegrounds were essentially negative. There was marked generalized lymphadenopathy. The lungs were clear to auscultation and percussion. A grade II apical systolic murmur was heard over the precordium. The abdomen was distended and had a doughy feeling. The tip of the spleen was palpable. The wrist, elbow, ankle and knee joints were swollen, hot and tender.

During the first admission complete blood counts revealed values of hemoglobin ranging from 59% to 70%. The white blood cell count varied from 13,650 to 17,850. The granulocytes predominated in all the differential counts. The urinalysis were repeatedly negative. A throat culture done on October 21, 1955 grew alpha and beta hemolytic streptococci. Subsequent repeated throat cultures grew only alpha hemolytic streptococci. Repeated blood cultures were negative. An anti-streptolysin titer test done on November 1, 1955 was 12 units. The blood serology (Kahn), sickle cell preparation, bone marrow studies, L. E. cell preparations, hemoglobin electrophoresis and tuberculin tests yielded negative results. A total serum protein of 6.3 grams/100 cc was obtained on 10/13/55. The albumen and globulin values were 3.3 grams/100 cc. and 3.0 grams/100 cc. respectively.

Electrocardiographic studies on October 10, 1955 showed a sinus tachycardia. A X-ray film of the chest done on October 10, 1955 was negative. On the same day X-ray films of the wrists and ankles revealed soft tissue swelling but no bone pathology.

The patient was admitted with the probable diagnosis of acute rheumatic fever, the possibility of rheumatoid disease and generalized lupus erythematosus to be ruled out. From October 9, 1955, the date of admission, until November 20, 1955 he received Meticorten 25 mg. daily. During the same period he received a wide-spectrum antibiotic. On November 14, 1955 fusiform swelling of the fingers was observed for the first time. On November 29, 1955 the patient was discharged moderately improved.

On October 12. 1956 the patient was admitted to the Department of Surgery of the San Juan City Hospital because of a traumatic complete fracture of the left femur. Since discharge he had been living in very poor condition, had not followed any treatment and his disease had progressed. Physical examination revealed a poorly nourished pale chronically ill child. The skin was shiny, thin and there was increase of body hair. There was marked generalized muscle atrophy. Examination of the heart and lungs was essentially negative. The liver and the spleen were not palpable. The abdomen had a doughy feeling. There was bilateral fusiform swelling of the fingers. The ankle, knee, wrist, elbow,

shoulder and temperomandibular joints were swollen and tender but had no increase in temperature or change in color.

The laboratory findings during this admission revealed a moderately severe microcytic, hypochromic anemia, a granulocytic leukocytosis and an elevated sedimentation rate.

He was treated with aspirin and penicillin. He spiked a rectal elevation of temperature up to 104°F., on several occasions. On October 22, 1956 he became afebrile and remained so until November 10, 1956 when he was discharged and referred to Crippled Children Clinic. Patient has failed to come to follow-up clinic.

Case III: A. M. C., a 9 year old white female admitted on August 27, 1956 with history of fever, abdominal pain, headache and arthralgia of four weeks duration. Two weeks prior to the onset of present illness she had suffered with sore throat and fever. Physical examination done on admission revealed a rectal temperature of 100°F. and a blood pressure of 120 80. The tonsils were hyperemic. The rest of the examination was negative except for swelling and tenderness of the right ankle, left knee, right elbow and the proximal interphalangeal joints.

The following laboratory studies revealed negative results: complete blood count, urinalysis, throat culture, antistreptolysin titers, sickle cell preparation, and the C-reactive protein. Total serum proteins done on August 17, 1956 and October 13, 1956 gave values of 7.85 grams 100 cc and 6.4 grams/100 cc respectively; the albumen-globulin ratio was inverted.

X-ray films of the hands and knees revealed no bone pathology. The electrocardiogram was negative.

During the hospitalization she complained of aching pain over the involved joints. She was treated with aspirin and discharged on October 15, 1956. Since then she has been followed in the Outpatient Department and has remained in remission.

Case IV: R. T. A., is a 2 year old colored male admitted on March 8, 1955 with the history of chronic bloody diarrhea and parasitosis of one year duration. Six months prior to admission he had developed inflammation of elbow, and knees, followed in a short time by fusiform swelling of the fingers.

Physical examination on admission revealed a pale, malnourished male. The rectal temperature was 99°F. The tongue was fiery red and smooth. The tonsils were injected and hypertrophied. The rest of the physical examination was negative except for swelling of wrists and ankles. There was bilateral fusiform swelling of the fingers. There was no increase in heat or redness over the involved joints.

Laboratory data revealed a microcytic hypochromic anemia. There was moderate leucocytosis with predominance of the granulocytic series. There was a constant eosinophilia. The urinalysis was negative. The sedimentation rate was 0. The total serum protein was 6.0 grams 100 cc., albumen was 3 grams 100 cc. Many trichuris and ascaris ova were seen in the stools. The throat culture was positive for B hemolytic streptococcus. Periarticular soft tissue swelling with atrophy of the osseous structure was seen in a X-ray examination of the knees, wrists and hands done on March 24, 1955.

Patient received treatment for the massive parasitosis and malnutrition and was discharged on April 11, 1955.

Case V: G. C. N. is a 2½ year old white female admitted on March 25, 1955. Prior to admission she had been hospitalized in the Army for 8 months. The diagnosis of juvenile rheumatoid arthritis was made for which she received cortisone therapy with some response. Two months prior to admission to our hospital she had a recurrence of spiking temperature, increased perspiration and generalized arthralgia.

Physical examination on admission revealed a rectal temperature of 103°F., a pulse of 160 beats per minute and respiration of 38 minute. The patient was well developed and poorly nourished. The tonsils were hypertrophied and injected. The rest of the examination was negative except for tenderness and swelling of the wrists and ankles and slight bilateral fusiform swelling of the fingers.

Laboratory tests revealed a hemoglobin of 63% and moderate granulocytic leucocytosis. The sedimentation rate was elevated. The albumin-globulin ratio was inverted. The sickle cell preparation was negative.

X-ray films of the hand revealed soft tissue swelling of the proximal interphalangeal articulations.

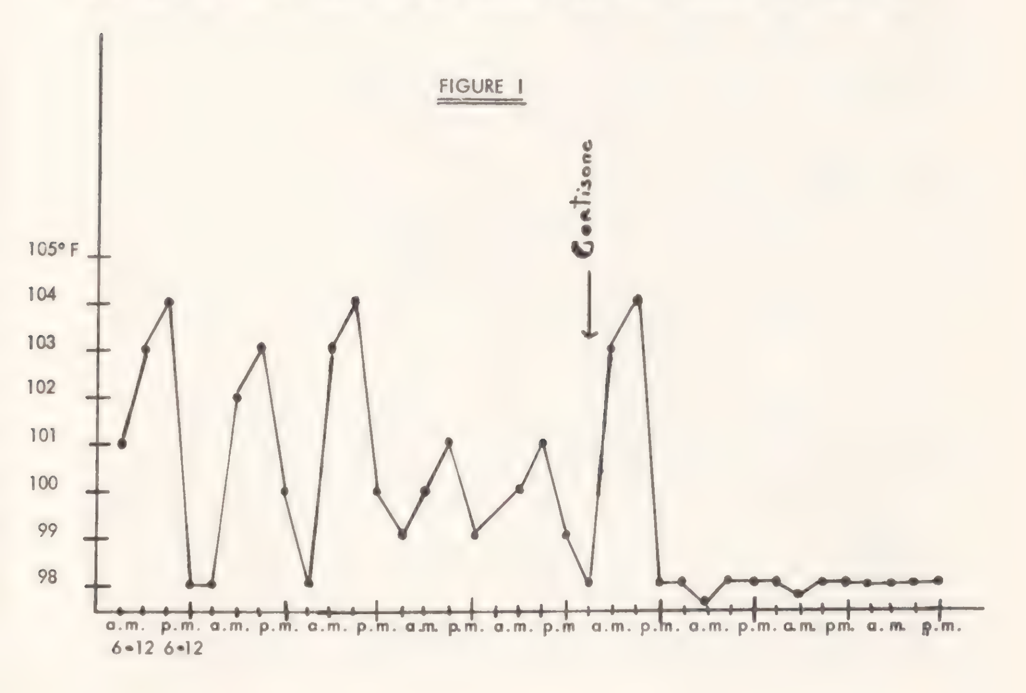
The patient received treatment with aspirin in a dose of 2½ grains every 4 hours. Two days later she became asymptomatic and remained so until discharged on April 6, 1955. On June 1, 1955 she was re-admitted because of an exacerbation of the disease. The sedimentation rate was elevated. An L. E. cell preparation was negative. A bone marrow study showed normal activity. X-ray films of the elbows revealed bilateral subchondral bone defects. From June 1, 1955 to September 1, 1955 she received 2½ grains of aspirin every four hours. Because of poor response to this treatment she was started on prednisone, 20 mg. daily on May 13, 1955. The dose was gradually tapered off until September 1, 1955 when she was discharged. She was referred to the Crippled Children Clinic.

DISCUSSION

Three of our patients were males. The age at onset of the disease ranged from 2 to 9 years. Two of the patients (Cases III and IV) followed the classical pattern of slow onset and progression characterized by mild systemic reaction. On the other hand, three patients had violent systemic reaction (I, II and V) and of these three patients two had rather late joint changes (I and II).

Arthralgia without local signs of inflammation is commonly an early manifestation. It can be transitory or migratory.[1] The order in which the joints are involved is extremely variable, but very often the first joints to be affected are the knees and ankles. It is interesting to point out that a single joint, most often a knee, may be affected long before other joints are involved.[2] Patient I and II presented swelling of the knees, ankles, elbows and wrists for at least 15 days before fusiform swelling of the fingers was noted. The first patient did not even show local signs of inflammation in the joints until one month after the onset of the disease.

Fever is a very striking feature of the disease and may be the only initial finding. It is usually of a high spiking type persisting for weeks and does not respond to salicylates or antibiotics.[1] Figure I illustrates part of the fever curve in the first patient. He had daily spikes of temperature, usually in the evening, for 47 days in spite of salicylates and antibiotic therapy. He became afebrile for the first time 24 hours after cortisone was started.



The presence of an erythematous maculopapular rash has been accepted as a dependable diagnostic sign in acute juvenile rheumatoid disease. It has been observed in 80% of the patients.[1] Patient I developed an urticarial rash on several occasions. At that time we interpreted it as an allergic response to the antibiotics. In retrospect, one wonders if the rash was really part of the clinical picture of juvenile rheumatoid disease.

Generalized lymph node enlargement is another common finding of the disease. The general incidence is 60%.[3] Involvement of the mesenteric lymph nodes may account for the severe abdominal pain of the acute cases. Cases I and II had marked generalized lymph node enlargement. In the first patient a lymph node biopsy was done to rule out malignancy.

About 15-30% of the patients with juvenile rheumatoid disease develop splenomegaly. Hepatomegaly is found in 12-23% of the cases.[3] The first patient in our series had at one time a palpable liver. Splenomegaly was found in only one patient (Case II).

Pericarditis is one of the most important visceral lesions. It is more commonly seen in children than in adults. Carditis is less frequent but may be the first manifestation of the disease. The cardiac changes tend to disappear gradually and completely.[1] On the other hand carditis is considered as one of the causes of death of patients with acute rheumatoid disease.[1] Two out of the five patients had involvement of the heart. Patient I had clinical, electrocardiographic and X-ray changes indicative of carditis. Patient II had electrocardiographic changes suggestive of pericarditis. Apparently neither had permanent heart damage.

Pleuritis and pneumonitis are also considered part of the clinical picture.[5,6] Only one of our patients, (Case I) had any pulmonary pathology.

Subcutaneous nodules are rarely seen in juvenile rheumatoid disease. Patient II developed a single subcutaneous nodule over the tibial area of the right leg. The nodule was removed surgically and the microscopic examination revealed an empty cystic structure lined by cuboidal cells.

General muscle and skin atrophy and profuse sweating were prominent signs in Patients I and II. Hyperpigmentation of the skin, liver palms and band-shaped keratitis, which can be seen in juvenile rheumatoid disease were not observed in our patients.

Radiographic changes in rheumatoid disease are not specific and may not appear for 1 to 2 years after the onset of joint inflammation except for soft tissue swelling. The first finding to appear and so the most helpful one in doubtful cases is generalized osteoporosis, most marked in those portions of the bone closest

to the affected joint. The later joint changes are progressive narrowing of the bone spaces, irregularities of the articular surfaces and cyst-like areas of bone destruction near the joint margin.[5]

Table I is a summary of the laboratory data obtained in the five patients. Two patients showed sedimentation rate values below 25 mm hr. One of these patients (Case III) had very little systemic manifestations of the disease. The other patient (Case IV) was admitted during a remission. Three patients had sedimentation rates (Cases I, II, V) close to 40 mm hr. and clinically they had marked systemic reaction. It is the general consensus of opinion that the sedimentation rate parallels closely the activity of the disease.

Leucocytosis ranging from 20,000 to 50,000 cells per cubic mm, with a predominance of the granulocytic series, is one of the most common findings in patients with active rheumatoid disease.[5] This is well illustrated in our small series. Patient III had a normal count which goes along with the mild systemic reaction she presented. Patient IV had a moderate granulocytic leukocytosis in spite of the fact that he was in remission. A throat culture in this patient grew B hemolytic streptococci which may account for the leukocytosis.

Anemia of the normocytic, slightly hypochromic type is a common complication in the course of active rheumatoid disease. The anemia does not respond to iron therapy and blood transfusions have proven of no benefit.[7] In a recent article[1] it is specifically stated that the anemia may become quite severe but that these patients should not be transfused unless the indications are imperative because they are usually prone to have transfusion reactions.

Four patients (I, II, III and V) had at some time during their illness a hemoglobin below 65% of normal. The first three patients developed marked microcytosis and hypochromia. Besides the rheumatoid disease, the deficiency of iron and the intestinal parasites influenced the hematological picture in these patients.

Changes in the serum proteins were observed in 4 cases (I, II, III and IV). The usual finding was an inversion of the albumin-globulin ratio.

Antistreptolysin titers have shown increased values in rheumatic fever, but normal or slightly elevated values in rheumatoid disease.[8] This test was done in only one patient (Case II) and a value of 125 u was obtained in spite of the fact that he had a positive throat culture for B hemolytic streptococcus. Two other patients (I, IV) had on one occasion a positive throat culture for B hemolytic streptococcus.

Agglutination tests were not done. These tests are all based

TABLE I

SUMMARY OF LABORATORY DATA

	Sedimentation Rate	C-Reactive Protein	W. B. C.	Granulocytes	Hemoglobin	Color Index	R. B. C. Morphology	Serum Protein T	Serum Protein A	Serum Protein G	L. E. Cell Prep.
A. L. D. Case I	25-32	N. D.	16.450-51,200	66-95%	36-62%	0.6-0.9	* M	8.25	3.88	4.37	Neg.
A. R. C. Case II	34-36.5	+4	13,650-17,850	64-85%	59-70%	0.6-0.9	* M	7.8	3.7	4.2	Neg.
A. M. C. Case III	14-16	0	6,300-8,500	49-70%	72%	0.9		7.85	3.7	4.15	Neg.
R. T. A. Case IV	0	N. D.	15,450-21,000	77-81%	45-67%	0.5-0.7	* M	6.0	3.0	3.0	N. D.
G. C. N. Case V	36-40	0	17.050	64%	63%	0.9		7.5	4.4	2.8	Neg.

* M — Microcytosis
N. D. — Not Done

on the fact that the serum of patients with rheumatoid disease is capable of agglutinating sensitized particulate bodies.[9]

Because of the variability of the clinical picture, rheumatoid disease must necessarily enter in the differential diagnosis of many important clinical entities.

Disseminated lupus erythematosus can mimic rheumatoid disease in all respects including the destructive joint changes.[3] The findings of L. E. cells and leukopenia favors the diagnosis of lupus. Pericarditis is more commonly seen in lupus. Splenomegaly is quite common in both diseases. We obtained negative L. E. cell preparations in the four patients, (I, II, III and V) in whom they were done.

Rheumatic fever is at times very difficult to differentiate. The presence of pancarditis, prolongation of the P-R interval in the electrocardiogram, high antistreptolysin titer, and a dramatic response of the polyarthritis to salicylates favor the diagnosis of rheumatic fever. In rheumatic fever the white blood cell count is seldom as high as it is seen in active juvenile rheumatoid disease.

Acute leukemia must be considered in the differential diagnosis of acute juvenile rheumatoid disease. Recently we admitted a five year old male with history of pain and local signs of inflammation over the left knee and right wrist of 5 days duration. There was also a history of spiking temperature for one month prior to admission. The diagnosis of acute rheumatoid disease was favored. He was started on prednisone and a wide spectrum antibiotic shortly after hospitalization but even on this therapy he continued spiking the temperature. Two months after admission he developed swelling and redness of both metatarsal regions. The clinical picture remained more or less consistent with juvenile rheumatoid arthritis until 3 months after admission when he developed purpura, splenomegaly, leukopenia and thrombocytopenia. A bone marrow aspiration at this point revealed blast cell leukemia and radiographic studies of the bone showed lesions compatible with acute leukemia. It is noteworthy that a previous bone marrow study had been negative for malignant cells.

Anaphylactoid purpura and tuberculosis can also present a clinical picture similar to rheumatoid disease.

Thus far, none of the forms of treatment used has been of any value in altering the course of juvenile rheumatoid disease. The conservative part of the treatment including adequate feeding and physical therapy is most important. Steroids should be reserved for patients seen early in their illness with severe constitutional reactions.

SUMMARY

Five cases of juvenile rheumatoid arthritis admitted over a three year period to the San Juan City Hospital have been reported. The salient clinical features of this disease, as seen in the pediatric group, were discussed.

The fact that the disease is most protean in its manifestations and can mimic other clinical conditions such as generalized lupus erythematosus, rheumatic fever, leukemia, anaphylactoid purpura and tuberculosis has been stressed.

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EDITORIAL

EL MONSTRUO QUE AMENAZA

La Asociación Médica de Puerto Rico se halla empeñada en tenaz lucha en contra de todo intento de destruir el libre ejercicio de la profesión, por entender que este libre ejercicio es el que hace posible el máximo beneficio para el enfermo.

Se pretende interferir con la libre selección del Médico. Se habla y se escribe subrepticiamente de que el gobierno debe ser responsable de la salud de todo (¡ !) ciudadano. Se planifica un programa de regionalización en el cual se desvirtúan los verdaderos conceptos del vocablo, y se plantean en efecto los comienzos de una medicina regimentada y socializada — un verdadero lobo disfrazado de abuela — incluyendo recomendaciones urgentes a nuestro gobierno de que utilicen los hospitales públicos para atender otros enfermos que no son los indigentes. Se extiende el monstruo, como la hidra de las siete cabezas, y aparecen, en rápida sucesión, planes en un Centro Médico para hacer un hospital privado administrado por la Escuela de Medicina, un informe de servicios médicos preparado por un grupo de expertos extranjeros y del patio, en que se habla de la conveniencia de utilizar los hospitales de gobierno para los pacientes privados, y, más recientemente, unos proyectos de ley preparados por la oficina del Contralor, con los que se pretende darle el santo y bueno a la admisión de pacientes privados en hospitales públicos.

Al presentar la Asociación Médica de Puerto Rico batalla frontal a todas esas ideas, se retira el monstruo, pero solo momentáneamente, en lo que su protoplasma se reorganiza, para poder hacer que germine una nueva cabeza.

Compañeros, ¡alerta!. Con el verbo, con la pluma, con la acción, con el precepto, con el ejemplo, aprestémonos a amputar toda nueva cabeza que plasmare el monstruo que amenaza.

IN MEMORIAM



Manuel Fernández Fuster, M.D.
(7 de enero de 1912 — 21 de enero de 1958)

De lo expresado por los que pertenecen a la Sección de Obstetricia y Ginecología citamos: "Compartimos un sentimiento que se ha ido fortaleciendo a través de los años que hemos estado con él; nos sentimos privilegiados por haber tenido la oportunidad de beneficiarnos de la inspiración que siempre nos brindó nuestro jefe. Esa inspiración seguirá siendo, entre todos nosotros, el estímulo orientador del Maestro."

Nos unimos a ellos en la dedicación de un pensamiento noble a quien honor merece: por el hombre, el compañero, el profesor, por el médico, por el doctor Manuel Fernández Fuster, q.e.p.d.

A. M. de Andino, Jr., M.D.

SECCION ADMINISTRATIVA

CARTA MENSUAL DEL PRESIDENTE

Principios de Etica: El Comité de Etica de nuestra Asociación, presidido por el doctor Luis A. Sanjurjo, desea traer a conocimiento de la matrícula, por nuestro conducto, las siguientes secciones de los Principios de Etica que gobiernan las actuaciones de los médicos:

"Section 5: A physician may choose whom he will serve. In an emergency, however, he should render service to the best of his ability. Having undertaken the care of a patient, he may not neglect him; and unless he has been discharged he may discontinue his services only after giving adequate notice. **He should not solicit patients."**

Section 6: A physician should not dispose of his services under terms or conditions which tend to interfere with or impair the free and complete exercise of his medical judgment and skill or tend to cause a deterioration of the quality of medical care."

Suplicamos a los compañeros asociados tomen debida nota de estas dos importantes disposiciones de nuestro Código de Etica, evitando así que el comité tenga que llamarles la atención sobre estos aspectos.

Queremos aprovechar la ocasión también para informar a la matrícula que la Cámara de Delegados de la Asociación Médica Americana, en reunión celebrada en junio de 1955, aprobó la Resolución Núm. 77, que dispone como sigue:
"ASSOCIATIONS BETWEEN DOCTORS OF MEDICINE AND OPTOMETRISTS ARE UNETHICAL".



Asamblea Anual - Asociación Médica del Distrito Oeste: Es con suma satisfacción que informamos a la matrícula que la asamblea anual de la Asociación Médica del Distrito Oeste, que preside el doctor J. Ramírez Ledesma, se celebrará en la Casa Rotaria de la ciudad de Mayagüez los días sábado 15 y domingo 16 de marzo, con un interesante programa administrativo y científico-social, que nos complacemos en reproducir a continuación:

Sábado, 15 de marzo de 1958 - 5:00 p. m., Casa Rotaria:

1. Apertura y dedicatoria del acto, J. Ramírez Ledesma, M.D.
2. Mensaje del Presidente de la Asociación Médica de P. R., Luis R. Guzmán-López, M.D.
3. Informe del Secretario, Ramón E. Ramírez, M.D.

4. Informe del Tesorero, Augusto Perea, M.D.
5. Informes de Comités
6. Asuntos a discutir
7. Elección de nueva directiva
8. Concierto, a cargo de la soprano señora Olga Iglesias, acompañada al piano por la señora H. Hutchinson.
9. Buffet, cortesía del señor Luis Garratón y A. H. Robins Co., Inc.
10. Demostración de sonido estereofónico de alta fidelidad, a cargo de los profesores Oscar Porrata Doria y Luis E. Fiol, del Colegio de Agricultura y Artes Mecánicas de Mayagüez.

Domingo, 16 de marzo - Casa Rotaria de Mayagüez, 9:00 a.m.

1. Evaluación de distintos métodos para el diagnóstico del Cáncer del Cérvix Uterino, Carlos Alemañy, M.D.
2. Choice and Complications of Anesthesia in Everyday Practice, Iván H. García, M.D.
3. Narcóticos: A Hazard to the Medical Profession, Lcdo. Pedro M. Vélez, Jr., Director de Investigaciones Fiscales.

   Receso de 15 minutos para tomar café.
4. Medical Applications of Radioisotopes, Angel A. Cintrón Rivera, M.D.
5. Clinico-pathological Conference, Donald Jutzy, M.D. and José A. de Jesús, M.D.
6. Almuerzo en compañía de las esposas, en el Yagüez Rifle, Pistol and Shotgun Club y toma de posesión de la nueva directiva.

   Orador del día: Hon. Guillermo Arbona, Secretario de Salud.

El programa oficial de esta asamblea circulará en el curso de esta semana. Si el mismo no llega a tiempo, considere ésta como su invitación para que asista a este importante acto científico, social y administrativo.

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Primer Curso Postgraduado - Año 1958: El doctor Ramón M. Suárez, presidente de la Junta de Cursos Postgraduados, desea informar a la matrícula, que el primer curso del año 1958 estará a cargo de los doctores Cecil J. Watson y Owen W. Wangensteen, profesores de Medicina y Cirugía, respectivamente, en la Universidad de Minnesota. El curso de los doctores Watson y Wangensteen dará comienzo el LUNES, 31 de marzo, y constará de las siguientes conferencias:

Lunes, Marzo 31

8:00 p.m. The Problem of Conservative Management of Peptic Ulcer, C. J. Watson

8:45 p.m. Studies on Etiology and Surgical Management of Peptic Ulcer, Owen W. Wangensteen.

Martes, Abril 1

8:00 p.m. Esophagitis and Cardiospasm, Owen W. Wangensteen.

8:45 p.m. Some clinical and fundamental phenomena related to disturbances of the serum proteins, Cecil J. Watson.

Miércoles, Abril 2

8:00 p.m. The diagnosis and treatment of functional bowel disturbances, Cecil J. Watson.

8:45 p.m. Intestinal Obstructions, Owen W. Wangensteen.

Jueves, Abril 3

8:00 p.m. Studies on the Etiology of Gallstones, Owen W. Wangensteen.

8:45 p.m. Recent advances in knowledge of the bile pigments and hemoglobin metabolism, Cecil J. Watson.

Viernes, Abril 4

8:00 p.m. Clinico-pathological Conference, Cecil J. Watson

8:45 p.m. Clinico-pathological Conference, Owen W. Wangensteen

Además de la innovación de presentar un internista y un cirujano de fama internacional, la Junta de Cursos Postgraduados ha designado a los siguientes compañeros para que inicien la discusión en las distintas noches: doctor Federico Hernández Morales, doctor Ramón A. Sifre, doctor José Noya Benítez y doctor Francisco Raffucci. El doctor Enrique Koppisch discutirá las conferencias clínico-patológicas.

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Asamblea Anual - 1958

El Comité Científico, bajo la presidencia del doctor Calixto A. Romero, ya ha empezado a trabajar en la organización del programa para la próxima asamblea anual de nuestra Asociación, a celebrarse del 18 al 22 de noviembre.

La Sección de Medicina Interna y la Sección de Pediatría han sometido ya sus candidatos para seleccionar el internista y el pediatra que habrán de participar en la asamblea. La Sección de Cirugía someterá sus candidatos próximamente.

Las sessiones clínicas de esta ocasión etarán a cargo de las siguientes instituciones: Hospital Auxilio Mutuo, Hospital de la Capital, Hospital San Patricio y Escuela de Medicina. Suplicamos a los compañeros directores de estas instituciones procedan a organizar sus correspondientes actos, de manera que sometan sus programas al Comité con la debida anticipación.

Los compañeros interesados en participar en la asamblea deberán comunicarse con el doctor Romero oportunamente y empezar a elaborar sus ponencias prontamente.

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Nuestra Felicitación

Deseamos extender nuestra más cordial felicitación a los compañeros dirigentes de la Sección de Pediatría y del Capítulo local del American College of Surgeons, por los magníficos actos de carácter científico-social que presentaron con motivo de sus respectivas asambleas celebradas durante el mes de febrero.

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Proyectos de Ley

Los proyectos de la Cámara números 343, 344 y 345 son, en esta sesión legislativa, los tres proyectos que instrumentan la creación de planes de seguro médico-quirúrgico voluntario, tipo Escudo Azul. Estos proyectos tienen el firme endoso de la Asociación Médica de Puerto Rico y es el deber de todos los compañeros acercarse a los señores legisladores personalmente, por carta o telegrama, favoreciendo estas medidas, que tanto contribuirán al bienestar de la clase media de nuestro país.

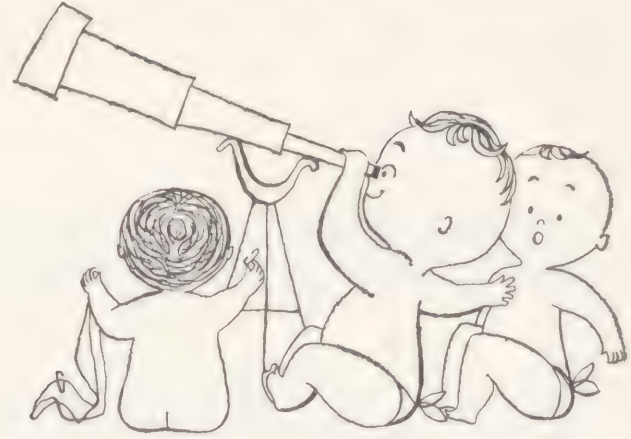
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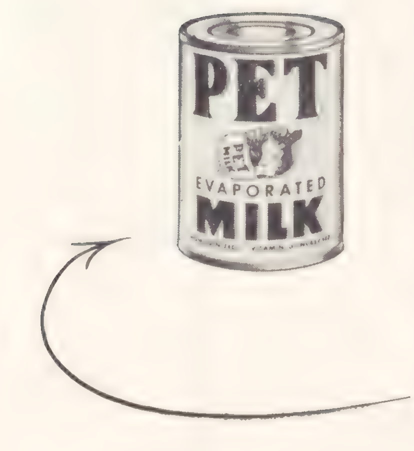
Encuesta Sobre Escudo Azul

Estamos transcribiendo al pie de esta página por tercera y última vez, el volante de consulta a la matrícula sobre Escudo Azul. Aún cuando la respuesta ha sido muy halagadora hasta la fecha, todavía quedan muchos compañeros que por una razón u otra no nos han brindado la cooperación que tan urgentemente solicita esta presidencia en relación al vital problema del Escudo Azul. Ruego encarecidamente a los compañeros que aún faltan por contestar, que se sirvan hacerlo a vuelta de correo.

Luis R. Guzmán-López, M.D.
Presidente







PET
PET MILK
EVAPORATED
MILK

ASOCIACION MEDICA DE PUERTO RICO

ASAMBLEA ANUAL

Noviembre 18-22, 1958

ASAMBLEA ANUAL

Asociación Médica de Puerto Rico

Noviembre 18-22, 1958

BOLETIN

DE LA

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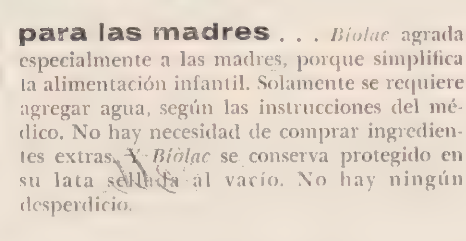
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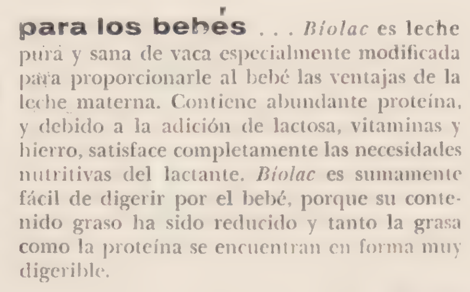
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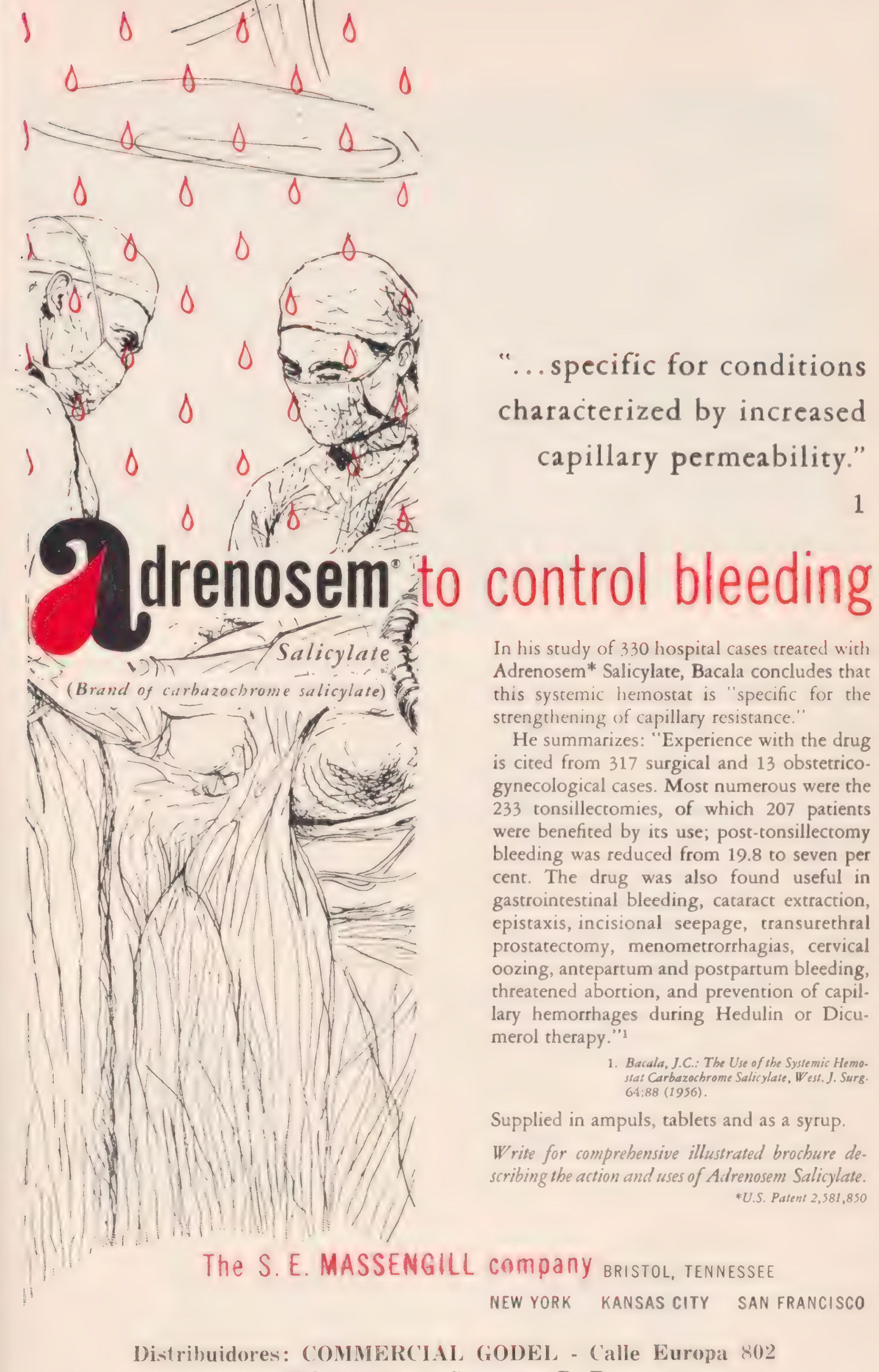
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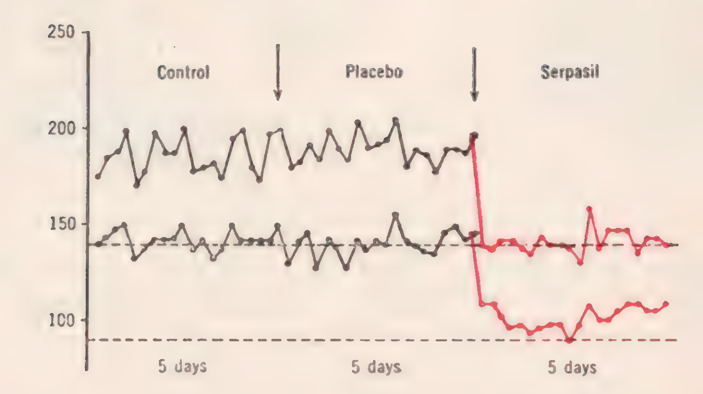


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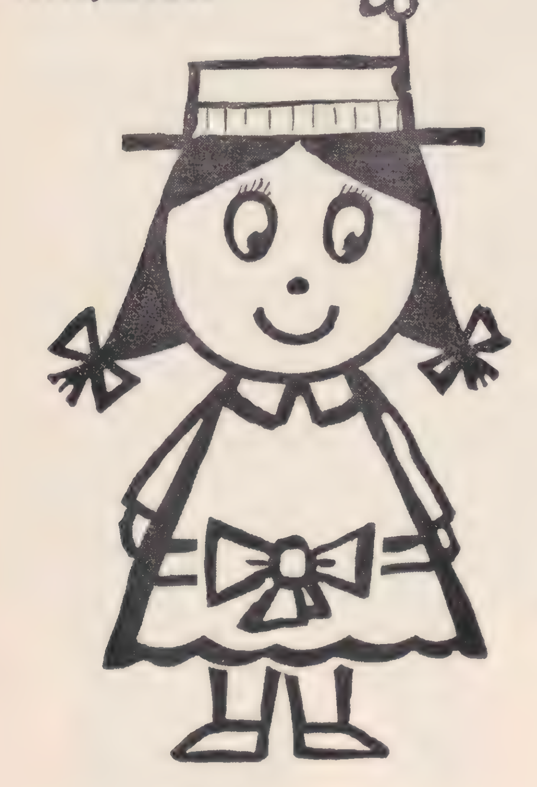


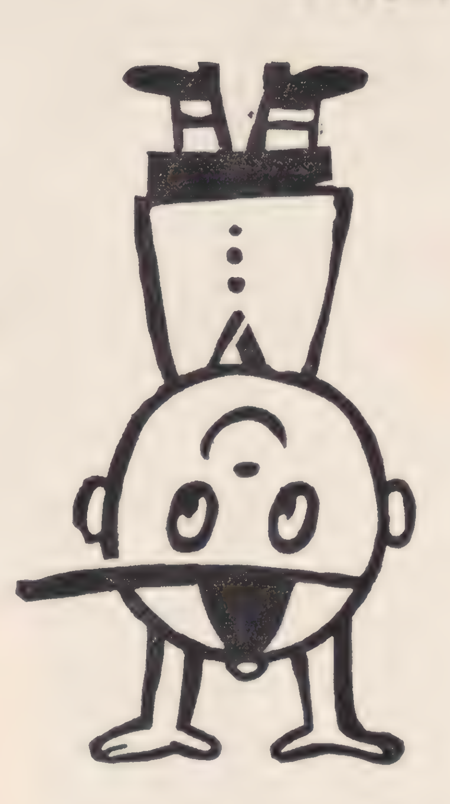
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BOLETIN

DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 50 MARZO, 1958 NO. 3

SCHISTOSOMIASIS MANSONI*

CLINICAL PATTERNS

*RAFAEL RODRIGUEZ-MOLINA, M.D.,*** *Dr. Med. Sc., F.A.C.P.*

The heavy and continued flow of Puerto Rican immigrants into the larger cities of northeastern United States during the past 15 years, has focused increased attention among physicians practising in these areas on the diagnosis, management and treatment of parasitic infections. Notably and foremost among these is Schistosomiasis Mansoni, constituting a particular problem, because it may be associated with fibrosis and/or cirrhosis of the liver, portal hypertension, splenomegaly with or without hypersplenism, and severe bleeding, frequently fatal, from ruptured gastroesophageal varices.

The organs most frequently affected as a result of infection with the blood fluke or flat worm, **Schistosoma mansoni,** are the colon, liver, spleen, rectum and lungs.

According to E. Koppisch,[1] Schistosomiasis mansoni may be classified into three stages: (1) an early stage of migration during which the cercarie are being carried by the blood to the liver, maturing into adult parasites within intrahepatic portal veins, (2) an intermediate stage during which ova are accumulating in various viscera, and (3) a late stage characterized by serious, irreversible, and permanent damage to organs, mainly through fibrosis.

In a general way the clinical manifestations of schistosomiasis may be correlated with anatomic changes resulting from the reaction of tissues to the parasites and their ova. Individuals affected with these worms may remain asymptomatic, or may present various clinical manifestations. The finding of schistosome ova in the stools of apparently healthy individuals is a relatively frequent one in endemic areas. Unlike the bacterial infections, the parasites do not multiply within the human host; thus, the

* Read at the American College of Physicians Post Convention Tour, Scientific Session, 15 April 1957, San Patricio VA Hospital, San Juan, Puerto Rico.

** Assistant Director, Professional Services for Research, San Patricio Veterans Administration Hospital, San Juan, Puerto Rico.

manifestations of the disease are dependent upon the intensity and frequency of exposure to infection.

Itching may or may not occur shortly after exposure, and urticaria is variable. Mild pyrexia may accompany urticaria. Anorexia, headache, generalized aches and pains, and mild diarrhea accompanied by abdomen discomfort may soon follow and last one to two weeks. These symptoms occur after invasion of the parasite and during the period of intravascular migration of the larvae or cercarie, as well as prior to the maturation of the worms. From 30 to 70 days following exposure, when the cercariae have become adult males and females and oviposition has occurred, more severe symptoms may appear in some cases. High fever (39° to 40°C), remittent in type and accompanied by chills, abdominal discomfort, and distention is present. Bloody, well formed stools, or bloody or mucous diarrhea may occur. A persistent dry cough ensues, and scattered fine or coarse rales are heard over the chest, suggesting bronchopneumonia. The peripheral blood presents an eosinophilic leukocytosis (14,000 to 20,000), and ova are usually found in the stools. The findings in the peripheral blood excepted, the clinical picture now resembles typhoid or paratyphoid fever.[2] But with the rarity of these infectious diseases either in Puerto Rico, New York City, Philadelphia, Baltimore, or in other large centers of population, the clinical picture when observed in young adult males recently arrived from endemic areas may resemble that of collagen disease or lymphoma, as the pyrexia may be similar to the Pels-Epstein type. The onset of the above picture may precede the finding of ova in the stools. Since 1847 the clinical manifestations in this, the acute, so called toxemic stage of the disease has been referred throughout the literature on diseases of the tropics as **Bilharzial fever,**[3,4,5,6,7] and has been considered by several authorities as an allergic reaction appearing between the fourth and sixth week following exposure and infection, constituting the acute stage of schistosomiasis. Fairley[8] is of the opinion that this early symptomatology is associated to the anaphylactoid reaction in the sensitized tissues of the host. He has classified the so called "toxic" substances responsible for the production of this state as:

(1) Specific glandular secretions produced by the cercariae or larval stage of the parasite, by the adult schistosomes and by the miracidia (embryonic forms in the eggs).

(2) Byproducts of metabolism of these forms.

(3) Excretions from adult worms to include catabolites of hemoglobin, such as bilharzial pigment.

In the human liver the male and female schistosomes worms live separately, but about four weeks after entering the body as

larval forms or cercariae, they become sufficiently mature to attain their period of sexual activity, join in pairs, and travel into the mesenteric veins forcing their way against the blood stream toward the small veins and venules of the submucosa of the large intestine. At this stage of migration it is probable that the toxic and antigenic material is liberated into the general circulation. According to Girges[3] this journey coincides with the presence of the acute or toxemic stage of the disease. At this point eosinophilia is observed, the complement fixation reaction and circumoval precipitin reaction become positive, and the anaphylactoid state, including urticaria, pyrexia, and pulmonary infiltration, may appear. The underlying cause of this allergic reaction is unknown, but it is probable that the body becomes sensitized to one or more of the schistosome substances mentioned by Fairley.[8]

With reference to the acute stage we wish to mention some of the highlights of the recent and excellent report by Díaz-Rivera and associates[9] comprising 12 cases occurring in young males who were thoroughly studied over a period of six years, following initial and repeated consecutive exposures and infection. This group of cases is believed to be the largest collection of clinical material ever reported in the acute phase of Manson's Schistosomiasis, presenting detailed and chronological picture of one of the aspects of the natural history of the disease, that is, the intracorporeal phase of the life cycle of the parasite. It was assumed that all individuals received massive infections with the parasites, giving rise to a closely similar, full blown picture in all cases. Clinically it was characterized by its sudden onset and by the explosiveness and severity of the constitutional manifestations, comprising shaking chills, spiking temperature up to 104 and 105°F, profuse diaphoresis, non-productive cough, generalized body aches and pain in the extremities, weakness, lassitude, nausea and vomiting, watery and bloody diarrhea, accompanied by tenesmus associated with generalized, crampy abdominal pain. Along with these there occurred moist rales over the lungs, hepatosplenomegaly, high eosinophilic leucocytosis, anemia, and a high serum globulin, with an increase of the gamma globulin fraction as shown by electrophoretic analysis. A clinical manifestation rarely mentioned in the literature, but present in all cases, was generalized lymphadenopathy.

After a variable period of pyrexia that lasted over 74 days in one case, the symptomatology gradually disappeared, improvement following defervescence, which subsided by lysis. No deaths occurred.

According to Díaz-Rivera,[9] this severe, though self-limited reaction associated with predominating constitutional manifestations

indistinguishable from an acute infectious disease, such as typhoid fever, is conditioned by the intensity of infection, degree of immunity and individual susceptibility. The severity of the symptomatology is dependent in part upon a state of tissue and organ hypersensitivity, the adult worms and their ova being the source of allergens.

The acute phase of Manson's Schistosomiasis has been previously described[2,10,11] in Puerto Rico and elsewhere, but is rarely observed by the practicing physician; moreover, in the majority of cases this type of reaction is mild and the clinical picture may amount to merely disturbances of general health. Loss of weight, lack of appetite, headache, abdominal and limb pains, mild pyrexial attacks, associated with possible transient urticarial eruption may then be the only clinical indications of a bilharzial infection. In Puerto Rico even this mild type of reaction is quite often either not observed by a physician or is diagnosed as something else.

When the initial infection is not severe, or when Antihelminthic treatment is given early the patient may recover rapidly and completely; but should the infection remain untreated, bloody diarrhea intermitent with constipation and accompanied by tenesmus and abdominal pain frequently occurs and lasts several months, the condition becoming chronic. The severity and duration of the gastro-intestinal symptoms depend on the intensity of the initial infection. In some untreated cases, however, several years may pass without the appearance of other indications of visceral disease, that is, if re-infection has not taken place. Should re-infection occur, however, fever and severe gastrointestinal symptoms may again ensue. Anemia, often mild, but at times severe enough to cause invalidism, may be present as the result of chronic blood loss. The anemia is hypochromic in type or normochromic.

When hepatosplenomegaly appears, the late manifestations of the disease frequently give rise to a new and ominous clinical picture. The clinical manifestations at this time resemble Banti's syndrome, with emaciation, hepatosplenomegaly, ascites, evidence of cirrhosis of the liver, and portal hypertension. Some cases develop hypersplenism associated with leukopenia, thrombocytopenia, and macrocytic anemia. However, the bone marrow is not megaloblastic. Liver function tests frequently indicate hepatocellular damage. Uncontrollable massive hematemesis from ruptured gastroesophageal varices is a frequent cause of death, and hepatic insufficiency is not uncommon. In Egypt rectal and colonic papillomas, producing prolapse of the rectum and intestinal obstruction, are late manifestations of the disease.

Recently, in a comparative study of portal and bilharzial cirrhosis comprising 91 male veterans in Puerto Rico evidence has been presented by Héctor Rodríguez and associates[12] to indicate the more frequent occurrence of portal hypertension associated with bilharzial hepatic fibrosis or cirrhosis, whereas fluid accumulation and symptoms and signs attributed to endocrine disturbances constituted a common feature of Laennec's cirrhosis. A higher incidence of symptoms associated with bleeding esophageal atrophy of testicles, palmar erythema, fetor hepaticus, retention of bromsulphalein and elevated serum bilirubin was encountered in the 69 individuals with portal cirrhosis. In the group of bilharzial cirrhosis comprising 22 cases, the main difference was the higher incidence of symptoms associated with bleeding esophageal varices, such as melena and hematemesis. The presence of hepatomegaly was the most common physical sign in both groups. Splenomegaly was encountered in 68% of the cases of schistosomal cirrhosis, but was present only in 33% of patients with portal cirrhosis. In addition, the spleen of the patients with bilharzial cirrhosis was much larger in size. Hyperglobulinemia and an abnormal cephalin—cholesterol flocculation were observed more often in the schistosomiasis group. In another series of 93 individuals including women and children with portal and bilharzial cirrhosis, again a higher incidence of symptoms associated with bleeding varices, such as melena and hematemesis was observed by Rodríguez[13] in the group of bilharzial hepatosplenomegaly. Esophagograms demonstrated esophageal varices in 67% of the group of Bilharzial cirrhosis and 25% in the portal cirrhosis group. The presenting symptom of an abdominal mass, in every case an enlarged spleen, was the most frequent complaint in 33 individuals comprising the schistosomiasis group, but was rarely encountered in the group of 60 cases with portal cirrhosis. On the other hand, loss of sexual libido, swelling of the legs or of abdomen were common symptoms in the Laennec's cirrhosis group, and rare ones in the bilharzial group. Other symptoms such as weakness, anorexia, fever, nausea and vomiting and abdominal pain showed no marked difference in the two groups. Regarding physical signs, hepatomegaly was the most common in both groups, but splenomegaly was encountered more often in schistosomal cirrhosis, than in subjects with portal cirrhosis. Splenomegaly in schistosomiasis was explained on the basis of mechanical factors, that is, intrahepatic periportal obstruction, rather than secondary to so-called toxic factors, for usually splenic enlargement is not associated with any specific lesion in that organ, such as the presence of ova, which is a rare finding.

With reference to liver function tests the results in the portal cirrhosis group presented the expected findings associated with severe hepatic insufficiency, most of the determinations showing a high incidence of abnormality. In bilharzial cirrhosis the liver function tests most often affected were those dealing with protein metabolism (cephalin-cholesterol flocculation, thymol turbidity, serum globulin) in addition to the bromsulphalein dye retention test and the alkaline phosphatase reaction. An interesting observation was that 40% of cases in the bilharzial group presented no impairment of liver function by laboratory tests, while in the portal cirrhosis group very few, or only 5% showed normal hepatic function tests.

In some cases, particularly in adolescents in whom irreversible vascular changes have occurred in the lungs, pulmonary hypertension associated with chronic cor pulmonale dominates the clinical picture, and another form of Ayerza's disease ensues which may overshadow the already present hepatic cirrhosis and portal hypertension.

In conclusion we can say, from our experience in Puerto Rico during the past 25 years, that clinical schistosomiasis in general may be roughly divided into three groups of cases:

First, and by far the largest group is made up of individuals who harbor living and dead **Schistosoma Mansoni** ova in the stools, but who are asymptomatic, as far as clinical signs of **S. Mansoni** infection are concerned. The second, a smaller group in which gastrointestinal manifestations, mainly episodes of diarrhea and abdominal pain, with or without blood or mucous, or tenesmus, comprise the clinical picture. The third, and fortunately a much smaller one, in which the individual presents evidence of irreversible and permanent visceral disease giving rise to a state of chronic invalidism associated with cirrhosis of the liver, portal hypertension, hypersplenism, and repeated episodes of hematemesis and melena, as result of bleeding from gastro-esophageal varices, frequently fatal.

The cases of acute schistosomiasis and of pulmonary hypertension have been rare in our experience.

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TETANUS AT THE PEDIATRICS DEPARTMENT OF THE SAN JUAN CITY HOSPITAL

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Our presentation will consist of a description of tetanus and a review of the cases of tetanus admitted to our Department during a period of two years: July, 1955, to June, 1957. The total number of admissions during that period was 3,619; 1,789 during the first year, and 1,830 in the second year. Thirty of these admissions were cases of tetanus, 0.8% of the total. Of these thirty cases, six, or 20%, were cases of tetanus neonatorum.

Tetanus is a non-contagious infectious disease caused by the action of the toxin produced by Clostridium tetani on the motor cells of the nervous system.[1] The disease was first described by Hippocrates, but its nature remained obscure until 1884, when Carlo and Rattone demonstrated its transmissibility. In 1885, Nicolaier observed the bacillus, and in 1889, Kitasato proved the inability of the bacillus to enter the blood stream, and that the symptoms were produced by the absorption of a toxin.[2]

Clostridium tetani is a gram-positive obligate anaerobe that under adverse conditions assumes the sporulating form, protecting itself from light and heat, thus being able to survive more than ten years. The bacilli as well as the spores are widely distributed in nature.[3]

Clostridium tetani produces tetanolysin and tetanospasmin. Tetanolysin has no clinical significance. Tetanospasmin reaches the central nervous system and becomes fixed to the ganglionic cells of the anterior horn of the medulla and the motor cranial nerves. This action renders the nervous system easily excitable, and even the slightest stimuli start dangerous spasms or tetanic convulsions. On the other hand, the characteristic muscular hypertonia is produced by a peripheral action of the toxin on the myoneural junction.[1] Thus, the disease may be generalized or localized. One of our patients had localized tetanus following trauma to the back. At first, the diagnosis was not suspected, but after other possibilities were ruled out, tetanus anti-toxin was given and the condition cleared in a week.

The site of entry of the organism is not always demonstrable and, though the disease usually follows puncture wounds, it may result from the most trivial scratch, insect bite, or superficial abrassion.[2] In newborns, the umbilical stump is usually the point of entrance for the organism, but circumcisions have also been blamed. Seven of our patients had no obvious site of infection.

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One patient had an infected tooth, and another had a chronic suppurative otitis media, both suspected sites of infection. In five of the six cases of tetanus neonatorum, the umbilical stump was found to be infected and in the sixth one, the focus of infection was not evident. The rest of the patients had lacerations, puncture wounds or burns preceding the onset of symptoms.

Five of the patients with tetanus neonatorum were delivered at home and one was delivered at this hospital and discharged on the fourth hospital day. He was admitted with a full blown picture of tetanus neonatorum when 11 days old, with symptoms of 24-hours duration and no obvious site of infection. Of the five delivered at home, one was transferred to a private hospital where the mother was to have a surgical intervention. The baby was a week old, an infected umbilicus was found and in 24 hours the patient has clinical evidence of tetanus.

Three positive cultures were obtained in our patients. The material for culture was taken from puncture wounds or lacerations in the sole of the foot in all three patients.

The incubation period is usually five to fourteen days.[3] In our patients, two cases had incubation time of less than five days; the rest ranged from five to fifteen days.

The period of time between the appearance of the symptoms and the occurrence of the first spasm has been called the onset time. According to Cole, the period of survival of the patient is roughly 3 times the onset time; less than 48 hours onset time means a very poor prognosis.[1] All of our patients developed symptoms within 48 hours prior to admission, and in twenty-nine of the patients the chief complaint was that of difficulty in taking food.

Fever is a bad prognostic sign. Temperatures up to 101º F. (rectal) were present in most of the patients. Five of them had temperatures above 102ºF. One of them had a pyelonephritis that explained the fever. Of the remaining four, three died: this supports the statement that fever is a bad prognostic sign.

The disease is most common between the ages of four to ten years and occurs more often in boys than in girls, in a proportion of 3:1.[3] Twenty one of our patients were males and nine were females. The youngest patient was five days old and the oldest, eleven years. Most of our patients were between the ages of five and ten years.

The treatment is directed toward the neutralization of the toxin with antitoxin, the prevention of convulsions, the prevention of complications and the maintenance of the patient's nutrition.[4] Our patients have received 60,000 units of tetanus antitoxin by the intravenous route and 40,000 units intramuscularly. 10,000

units of antitoxin were injected around the site of infection in patients where the focus was evident and surgical debridement was done. Penicillin, in doses of 400,000 to 800,000 units, was given daily. Penicillin inhibits the growth of the organism and serves as prophylaxis for secondary infections. Sulfonamides, to a lesser extent, also inhibit the growth of the organism.[5] Barbiturates are widely used for sedation, and sodium phenobarbital appears to be the drug of choice in treating tetanus neonatorum.[2] Dosage varies with the severity of the disease and the presence or absence of convulsions, but the initial doses are over the usual hypnotic dose of 3 mgm. per kilogram of body weight.

Other drugs used are: chloral hydrate, paraldehyde,[6] magnesium sulfate, avertin,[7] mephenesin,[1] promazine and clorpromazine. The last two are to be used in conjunction with barbiturates or mephenesin as they enhance the relaxant effects of those drugs without circulatory or respiratory depression. Curare has been used for the maintenance of full relaxation after the use of barbiturates or mephenesin. Muscular relaxants, as curare and avertin, have been preferred by Roseman in Louisville on the basis that the toxin has no deleterious effects on the brain.[8] Their use requires an extremely rigid supervision, as respiration may be depressed for days. The use of a respirator may be necessary in some cases.

In our patients sedation was accomplished with sodium phenobarbital. Some patients required up to two grains of phenobarbital on occasions where rigidity was marked. Nine of the patients received chlorpromazine, and one, promazine. Doses were in the neighborhood of 12.5 mgm. every six hours. Patients receiving this medication were relaxed promptly and satisfactorily, and only one of the patients had convulsions. They also required smaller doses of barbiturates.

A total of nine of our patients had convulsions, six of them before hospitalization, and three during their hospital stay. Only one patient required a tracheotomy.

The average hospital stay was 15.8 days; the longest 35 days, and the shortest two days (a patient with tetanus neonatorum that died).

The mortality rate throughout the world is 40-50%. Deaths are more frequent among newborns, over 75% mortality is reported of patients with tetanus neonatorum.[1] The factors influencing mortality are: convulsions, respiratory complications (mainly pneumonias and atelectasis), incubation periods of less than five days, age, and the general condition of the patient. In our patients there were four deaths: 13.33%. Of those, three were cases of tetanus neonatorum, to make 75% of the total deaths and 50%

mortality for tetanus neonatorum. The three patients with tetanus neonatorum died after one week in the hospital, and apparently with very shallow respirations, which were thought to be due to oversedation. No autopsies were done. The cause of death in tetanus neonatorum is unknown. The other patient that died was a boy five years old, who had first, second, and third degree burns in both upper extremities three days prior to admission. Of the four deaths, two were patients with incubation periods of less than five days.

As a final statement, it will be said that the rate of decrease of tetanus is proportional to the extent of tetanus immunization. Continued childhood vaccination will result in tetanus remaining as a disease of adults.[9]

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ROTURA TRAUMÁTICA DEL BRONQUIO FUENTE IZQUIERDO
(BRONCOPLASTIA CORRECTIVA)

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La rotura traumática de un bronquio es una complicación frecuente de los traumatismos por compresión del pecho. Desde hace alrededor de 50 años se conoce el cuadro clínico de estos traumatismos que conllevan una mortalidad inmediata muy alta. De alrededor de 50 casos que han sido informados en la literatura, la mitad de ellos murieron debido directamente a la patología envuelta.

La rotura total del bronquio fuente va seguida generalmente de neumotórax y o neumohemotórax con enfisema mediastínico. El pulmón donde ha ocurrido el neumotórax se encuentra en un estado de atelectasia masiva. Si sobrevive el paciente, el bronquio se sella por fibrosis dejando una estrechez total que puede medir hasta cerca de dos pulgadas como en el caso que vamos a informar.

Es curioso notar que el pulmón atelectático puede permanecer muchos años en este estado, y al repararse el bronquio re-expander con un sistema bronquial como antes de haber sufrido el traumatismo; en otras palabras, no desarrolla bronquiectasia próxima a la obstrucción. Si por el contrario, la obstrucción es parcial y ocurren infecciones próximas a ésta, desarróllanse entonces supuraciones bronquiales con destrucción de su arquitectura, fibrosis y abcesos pulmonares. Con la subsecuente pérdida de función pulmonar, la reparación del bronquio se hace impráctica y más bien existe entonces la indicación para la resección pulmonar.

No fué hasta que la cirugía torácica adquirió el auge que tiene hoy día, con los refinamientos en técnica, conocimientos de fisiología pulmonar, y el advenimiento de la anestesia endotraqueal, que se pensó en la broncoplastia reconstructiva con miras a preservar tejido y función pulmonar. La primera broncoplastia en un caso traumático con excisión de la estrechez y anastomosis término-terminal de bronquio fué publicada por J. L. Grifith en el 1949. Desde entonces los trabajos de Paulson,[3] de Gebahuer y las interesantes publicaciones de Samson y Mahaffey[1] nos han demostrado la importancia de la reconstrucción bronquial desde el punto de vista de reconquista de función pulmonar. Nos han traído también la confirmación de los conceptos ya expuestos por Croxatto y La-

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nari[2] en relación a la patogénesis de la bronquiectasia en las estenosis bronquiales.

Es nuestro interés informar un caso de reconstrucción de bronquio por estrechez total del mismo después de un traumatismo de pecho ocurrido 8 meses antes. En este caso no solamente se obuvo re-expansión de un pulmón sino que el paciente, según los estudios de función pulmonar antes y después de la operación, ganó también en este respecto.

INFORME DEL CASO

Se trata de un joven blanco de 24 años de edad admitido a la Clínica Antillas en noviembre de 1956 quejándose de malestar en el hemitórax izquierdo y de tos seca.

La historia de su enfermedad comienza el día 7 de junio de 1956 cuando el paciente fué atrapado por un camión que retrocedía apretando su lado izquierdo contra una pared mientras lo empujaba por el lado derecho del tórax. El paciente perdió el conocimiento y fué llevado a una clínica donde al tomársele radiografías se le encontró un neumotórax bajo tensión en el lado izquierdo. Se le colocó un tubo de drenaje intercostal y se refirió a una clínica en Santurce donde fue tratado con antibióticos. Al cabo de cinco días se le repitieron las radiografías y se encontró que las estructuras mediastinales habían vuelto al centro. Había una opacidad en el lado izquierdo del pecho, el enfisema subcutáneo había desaparecido considerablemente, por lo que se le removió el drenaje intercostal. El paciente, mejorado de su condición, pudo ser examinado mejor radiológicamente y se descubrió una fractura de la primera costilla del lado derecho. Además se le notó un síndrome de Horner del lado izquierdo que no había presentado antes. Al examen neurológico llegóse a la conclusión que éste probablemente se debía a un hematoma en la región prevertebral cervicotorácica. Durante esta hospitalización se le hicieron repetidas broncoscopías. El día 3 de julio de 1956 se le practicó una broncografía en la cual se confirmaron los hallazgos broncoscópicos de obstrucción total del bronquio fuente izquierdo media pulgada por debajo de la carina principal.

El paciente fué dado de alta el 2 de agosto de 1956 con los siguientes diagnósticos: 1—fractura del bronquio principal izquierdo con atelectasia total del pulmón izquierdo. 2—fractura de la primera costilla lado derecho. 3—síndrome de Horner lado izquierdo.

Ya en la Clínica Antillas se le hicieron tomografías y espirometrías encontrándose una reserva ventilatoria de 8.4. Los estudios espirométricos no demostraron déficit de oxígeno a pesar de haber una disminución moderada en la función pulmonar con un factor de broncoespasmo de 24.8 litros.

Operación

El 23 de enero de 1957 se le practicó una toracotomía exploradora. Se encontró el pulmón izquierdo totalmente atelectático. Se disecaron los extremos proximal y distal del bronquio izquierdo los cuales estaban separados cerca de dos pulgadas uno del otro. El segmento distal después de haberse limpiado aparecía más pequeño que el segmento proximal, el cual estaba en un plano profundo por detrás de la aorta. Al abrirse el bronquio se pudo aspirar una gran cantidad de mucosidad transparente del pulmón atelectático. Se introdujo entonces un catéter en el bronquio y se insufló aire a través de éste en el pulmón atelectático notándose que volvía a re-expandirse adoptando un color rosado y una sensación normal al tacto. Sin movilizar la aorta se decidió hacer una anastomosis término terminal. La parte anterior del bronquio se aproximó con puntos de seda 30 continuados y la parte membranácea con puntos interrumpidos. Después de terminar la anastomosis el anestesiólogo infló el pulmón a presión y se colocaron dos tubos de drenaje, uno superior y uno inferior y se cerró la herida en planos.

A los 7 días se le practicó una broncoscopía y se encontró que la anastomosis estaba cicatrizando bien. El paciente tuvo fiebre postoperatoriamente y fué tratado con acromicina y succiones endotraqueales por catéter. Las radiografías postoperatorias demostraron el pulmón bien aereado.

En febrero de 1957 se le hizo una broncografía donde se demostró una ligera disminución de diámetro en el sitio de la anastomosis. Como el paciente desarrolló sibilancia se le hizo una broncoscopía en abril de 1957 encontrándose tejido de granulación en la anastomosis bronquial. Se le sacó el tejido de granulación y 4 puntos de seda, con lo que desapareció el síndrome asmatoide. Espirometría hecha en marzo de 1957 demostró una reserva ventilatoria de 10, una disminución del factor broncoespástico a 5.4 litros. La impresión del consultor fué que se observaba una mejoría notable en la función pulmonar después de la broncoplastia y que probablemente mejoraría aun más con el tiempo. El paciente fué dado de alta para verse cada seis meses y seguírsele haciendo pruebas de función pulmonar.



A

Roentgenograma preoperatorio demostrando: A) Opacidad total del lado izquierdo con desplazamiento del mediastino hacia ese lado. Lipiodol en el bronquio fuente izquierdo que termina abruptamente ½ pulgada por debajo de la carina.





B C

Roentgenogramas post-operatorios demostrando: B) La expansión del pulmón izquierdo, regreso del mediastino al centro y descenso del diafragma izquierdo después de la broncoplastia izquierda. C) Broncografía post-operatoria demostrando árbol bronquial esencialmente negativo después de estar 8 meses atelectático.

RESUMEN

Hemos presentado un caso de rotura traumática del bronquio fuente izquierdo debido a un traumatismo por compresión de pecho.

Estudios preoperatorios de broncoscopía e instilación con lipiodol confirmaron la obstrucción total del bronquio.

Estudios espirométricos demostraron una disminución de la reserva ventilatoria de 8.4 que subió a 10 después de la reconstrucción.

Ha sido muy curioso notar que preoperatoriamente el factor broncoespástico era de 30.8 LO_2 mientras que después de la reparación bronquial bajó a 4.0 LO_2. Aparentemente el estado de dilatación constante del árbol bronquial por la acumulación de mucosidad por detrás de la obstrucción produce una serie de estímulos que se manifiestan reflejamente en el pulmón sano disminuyendo la ventilación en cerca de 24 LO_2.

Estudios broncoscópicos y broncográficos posteriores a la reparación hecha 8 meses después de sufrido el traumatismo demuestran reexpansión pulmonar perfecta y árbol bronquial normal.

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SUMMARY

A case of traumatic rupture of the left main bronchus is presented.

Preoperative studies, bronchoscopy and lipiodol instillation confirmed the above findings.

Spirometric studies showed diminution of ventilatory reserve to 8.4 which raised to 10 after end to end anastomosis of the ruptured bronchus.

It is of interest that preoperatively the broncho-spastic factor was 30.8 LO_2 while after the end to end repair it went down to 4.6 LO_2 which makes us think that bronchodilation in the atelectatic lung from accumulation of secretions produced reflex contralateral bronchospasm.

Bronchoscopy and bronchography after end to end anastomosis performed 8 months after the trauma, showed full reexpansion of the atelectatic lung and a normal bronchial tree with no evidence of bronchiectasis.

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MODERN CONCEPTS IN THE MANAGEMENT OF CLEFT PALATE

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INTRODUCTION

One of the most controversial problems in the field of Plastic Surgery today is the management of the cleft palate. Different medical centers throughout the country and abroad have different ideas on how to approach this problem.

In the past, the rationale for the repair of cleft palate was based on considering the problem as only that of closing the defect. Little consideration was given to the dynamics of the adjacent musculature, to the bony frame around the defect, or to the growth potential. One of the modern tendencies in the approach of this problem, is to consider the structures involved not static masses, but rather as growing structures. To this concept, I will limit myself.

Surgery in the past tended to interfere with the normal developmental changes and growth through interference with blood supply, scar formation and destruction or damage of growth centers. Surgery at the present tends to help in directing the natural growth processes into proper lines by the establishment of muscle balance across the defect.

EMBRYOLOGY OF THE LIP AND PALATE

Amongst the numerous theories that have been advanced in trying to explain the reason for cleft palate, two have been brought forth in trying to explain the pathological embriogenesis responsible for the cleft lip and palate.

(1) The so called "classical" hypothesis described by the embryologists Arey and Patton. They state that the anomaly is the result of fusion or incomplete fusion of the face buds in the case of the cleft lip, and the palatine processes in the case of the cleft palate. This hypothesis appears plausible to a degree especially in explaining total clefts. However, the classical approach fails to explain the simple or partial cleft (one will have to postulate that the fusion occurred selectively in the mid portion but not anteriorly or posteriorly.) Also left unexplained is the fact that should the defective process be completed at so early a stage, why greater defects of nearby organs did not occur and that the defect remained so localized.

(2) The other or so called "modern" hypothesis has been presented mainly by Fleishman. The men who advocate this theory

feel that the fusion responsible for the production of the clefts was not a process that occurred as such. The defect, they think, is due to failure of mesodermal tissue invasion into the epithelial wall, either by:

(1) Preventive action of the epithelium or (2) by poor mesodermal tissue quality as it invaded, with subsequent atrophy and separation. In any event there seems to be a persistence of an epithelial wall.

FUNCTIONAL ANALYSIS OF THE SURGICAL MANAGEMENT

In order to obtain physiologic closure of the cleft it is necessary to consider the muscular forces operating in the region of the pharynx, the palate and the lip. Failure of the hard and soft palate to unite in the midline produces a marked disturbance in the interplay and balance between the involved muscle groups. The pulling action of these muscles around the cleft accounts for the greater distance between the pterygoid plates, the consequent increased lateral width of the nasopharynx, the separation of the tuberosities of the maxilla and the width of the cleft itself. It has been proved by numerous investigators that the lateral dimensions of the nasopharynx are greater in the cleft palate individual than in the normal. The several muscles groups to be considered are: around the mouth, the orbicularis oris, followed posteriorly as an almost interlocking chain by the masseter and succinator, the levator and tensor veli palatini at the junction of the hard and soft palate, the lateral and middle pterygoid muscles, the glosso-pharingeus, palato-pharingeous, and the constrictor pharingeus muscle.

Last, but perhaps most important is the tongue, a powerful muscular organ which is actively and intimately related to the cleft in the palate from early intrauterine life. The tongue can rest in the floor of the mouth or can extend into the nasal cavity or nasopharynx. During swallowing and other forceful actions, the tongue plunges into the cleft to displace the palatal processes, thus widening the cleft and the nasopharynx.

It has been observed in cases of the Pierre Robin syndrome in which there is micrognathism, ptosis of the tongue and cleft palate, that the width of the cleft was found to decrease. This is explained by the fact that with the downward and forward growth of the mandible, the tongue assumed a more normal position in the floor of the mouth, no longer occupying the floor of nose to the same previous extent.

The several muscles groups exert a pulling action which is unopposed by the opposite muscles. Antagonism between paired

symmetrical muscles does not exist; nor opposed tension of each lateral group, which contributes to the divergence of the surrounding bony frame.

SURGICAL TECHNIQUE

If we consider the cleft as an open wound, it follows that by approximating the ends, the middle has the tendency to come together. Early closure of the cleft lip tends to mold the face into a more acceptable form. A more favorably alignment of the osseous segments of the palate follows the reestablishment of the lost balance forces. Since repair of the lip alters the configuration of the skeletal frame in the anterior segment of the cleft, it is reasonable that repair of the soft palate will achieve the same beneficial result in the region of the nasopharynx, which is none other than the posterior end of the wound. It has been observed that once the lip and posterior palate are closed, the lateral width of the cleft in the region of the hard palate (which is equivalent to the middle section of the wound) decreases. The eventual repair of the hard palate will be greatly facilitated at a later date.

Since the principles governing this surgical type of repair does not involve fracturing of the bone, or undue introduction of scar tissue, or severance of blood supply or damage of the growth centers of the structures involved, a better physiologic result be obtained. Also by bringing structures of mesodermal origin across the defect, and at the same time keeping nasal mucosa in the nose and oral mucosa in the mouth it helps to prevent diminution or arrest of growth of the structures involved. One of the principal features of this procedure is that it establishes normal balance where abnormal muscle tension was operating before, and that further growth and development of the pharynx and palate must follow the pattern imposed by the new muscular function.

It is observed in these cases that following the closure of the cleft lip the palatal processes converge toward the midline, and the Vomer bone deflects in a horizontal plane to its point of attachment to the palatal process (in case of unilateral clefts.) The greatest amount of narrowing was observed in the anterior alveolar region of the cleft. This molding action continued for months after lip closure. It is also noticed in these cases, that the nasal septum tends to righten itself from a previous almost horizontal position. After the closure of the posterior palate, overlapping alveolar segments anteriorly tend to undergo gradual reduction, as the maxillary segments increase in its anterior posterior length. It has also been observed that besides the undisturbed (lateral and anterio-posterior) growth of the palate, cra-

nium and face, the downward and forward migration of the floor of the nose with the rightening and elongation of the nasal septum, have proceeded in an uninterrupted fashion and in a pattern consistent with the behavior of normal children during this age.

ROLE OF THE ORTHODONTIST IN THE CLEFT PALATE TEAM

I believe that at this point of our discussion the role of the orthodontist as a member of the cleft palate team together with the Plastic Surgeon. Psychiatrist, Pediatrician, Social Worker and Speech Therapist should be mentioned. Orthodontic therapy is generally limited to the movement of teeth within the alveolar processes.

In the treatment of the child with cleft palate another possibility exists. It is sometimes possible to reposition entire bony fragments in the same way as an Orthopedic Surgeon. Orthodontic treatment may reposition the palatal processes by the expansion and rotation of the maxillary bone into a more desirable relationship. Many times these results are difficult to obtain because the scar tissue produced by previous surgical procedures prevents the mobilization of the segments. The age element is also a very important factor to the Orthodontist. The zygomatic-maxillary suture offers a point of rotation for the repositioning of the palatal process. Obviously, it will seem that the best result can be achieved while this suture is not yet calcified but sufficiently plastic to allow for the realignment of the maxillary segments. Another advantage of early Orthodontic treatment is that the unlocking of impacted palatal segments allows for the normal expression of alveolar growth and the eruption of teeth into a normal oral environment.

In summary; Orthodontic therapy in a child with cleft lip and palate permits the expansion and rotation of maxillary segments. Malposed and overlapping palatal processes serve to obstruct the nasal cavity and prevent the normal eruption and growth of teeth. Restoration of a more normal palatal architecture is conducive to normal growth and function.

SUBMUCOUS CLEFT PALATE

Another condition worthwhile mentioning in the discussion of cleft palate and too often omitted is that of submucous cleft palate. This condition has been described as a congenital deformity in which there is imperfect muscular union across the velum. The palate is short and the velopharyngeal closure imcompetent, so

that speech is nasal in quality and may even be unintelligible. However, speech is usually intelligible.

Symptoms necessary for diagnosis are:

(1) Patients are unable to hiss, whistle or gargle.

Physical examination will reveal a short palate, an uvula which is often bifid, and a gutter is noted in the midline of the velum. Transillumination or palpation shows an absence of muscle union in the midline and a notch in the hard palate. Similar conditions that may give similar symptoms and that must be ruled out before the diagnosis is made, are:

Congenital short palate, cerebral agenesis, paralysis of the palate, "Tonsillectomy Palate", functional rhinolalia following adenoidectomy.

Surgery consists of excision of the submucous portion of the cleft and a V-Y retroposition of the soft palate carried out in one stage. Speech therapy should be undertaken only if normal speech has not developed three months after operation. Surgical treatment is advised at or about two years of age when the diagnosis is made in infancy.

THE TREATMENT OF ACUTE OTITIS EXTERNA WITH FURACIN AND FURACIN SPECIAL EAR SOLUTION*

*MIGUEL ALONSO, M.D.***
Santurce, Puerto Rico

The treatment of acute infections of the external ear and canal still presents many problems. The choice of the proper chemotherapeutic agent as has been emphasized by Senturia and Broh Kahn,[1] Depends on the causative agent, —be it bacterial or fungi.

Otitis externa may be divided into the diffuse and the circumscribed types. The circumscribed otitis externa is an active pyogenic infection, usually caused by gram positive cocci. staphylococci and streptococci predominating and as a rule associated with furunculosis, swelling, redness and pain, and in some cases even a febrile reaction. The diffuse otitis externa is usually associated with the presence of gram negative bacillus, the most frequent offender being the pseudomonas aeroginosa. Occasionally the bacillus proteus is found. In less than 10% of the cases we have studied, fungi were identified as the causative agents. These findings compare with the incidence of fungi reported by the author in a previous report.[2]

The present report deals with two series of patients treated separately and at different seasons of the year with Furacin Ear Solution and a Furacin special ear solution whose formula is:

Furacin	0.2%
Orthochloro Mercuri Phenol	1.600
Benzocaine	3%
Polyethylene Glycol Base	9.5

Only cases exhibiting the signs and symptoms of an acute otitis externa are included in this report. A total of 86 patients were seen. 40 patients were treated with Furacin Special Ear Solution and 46 with the regular Furacin Ear Solution. For the purpose of discussion this report is divided into two parts. The study was done at two different seasons of the year and again the therapeutic agent in the first group was slightly different from that used in the second group. All patients treated in these two groups were seen in the acute phase of the infection and had not received previous therapy during the present episode for which they presented themselves for treatment.

The first group was treated during the Summer months, from July 1956 to the end of October 1956. These are the hot weather

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* Product of Eaton Laboratories, Norwich, New York.

** From the Department of Otolaryngology of the School of Medicine of the University of Puerto Rico.

months when people frequent the beaches and swimming pools, the humidity is high, and the incidence of infections of the external ear and canal is high. This group comprises the study of 40 cases of acute otitis externa.

The ages of this group ranged from 5 to 82 years. Ten patients were under 20 years of age.

METHODS OF STUDY

The procedure followed in all cases in this study was to take two smears from each infected ear or ears to be sent to the laboratory to be cultured for bacteria and fungi separately.

The treatment consisted in wiping the ear canal thoroughly with a cottton applicator or preferably cleaning it with a suction tip until completely clean and dry whenever possible. No local antiseptics were used. A cotton wick impregnated in the modified furacin solution was inserted in the ear canal, and instructions given to the patient to place two or three drops of the solution in the ear so that the cotton wick would be saturated with the modified furacin solution at all times. The patient was also instructed to apply heat and take sedatives for the pain.

The patients were seen daily or every other day, depending on the discomfort and severity of the infection. If the condition did not clear in ten days, the treatment was discontinued.

The culture revealed the following results in order of decreasing frequency.

1. Pseudomonas Aeroginosa	14
2. Hemolytic Staphylococcus Aureus	12
3. Aerobacter Aerogenes	7
4. Diphtheroids	7
5. Bacillus Proteus	3
6. Enterococcus	3
7. Escherichia Coli	1

Fungi were present in 6 cases; Aspergillus Niger in 5 cases and a Penicillum in 1 case.

The ears were found normal in the great majority of cases after 7 days or less of treatment. Only 4 patients necessitated 8, 9, or 10 days for the ears to return to a normal state.

Six patients, or 16%, developed a definite allergy of the ear and canal as manifested by marked irritation with the formation of discrete vesicles on and around the pre and post auricular region, associated with oozing of the skin and itching. In these cases the modified Furacin solution was discontinued and adequate antiallergic therapy instituted.

It is interesting to point out that both the Gram Positive and Gram Negative group responded equally well to the modified Furacin solution. The same holds true for the 6 cases of fungi.

The Gram Negative group of bacteria in pure or mixed cultures were identified in 22 instances. The Gram Positive in 14 and Fungi in 6. Two patients failed to show growth of bacteria or Fungi.

The second group of cases which comprised 46 patients was studied between the months of December 1956 and May 1957. The incidence of acute otitis externa during these months is not as high as during the summer months.

The ages of these patients ranged from 5 months to 72 years of age. Seven patients were under 20 years of age.

The same routine was followed as in the previously described group. Two smears were taken from each affected ear, to be cultured, one for bacteria and the other for fungi. After thoroughly cleaning the ear by dry wiping with cotton applicator or with a suction tip, a cotton wick impregnated in the Furacin solution was inserted in the canal and instructions given to the patient to put two or three drops of the solution in the ear four or five times during the day. The patients were told to apply heat to the involved ears and to take sedatives whenever necessary. The ears were inspected every day in some cases, specially at the onset of treatment or every other day. If the condition did not clear in ten days the treatment was discontinued.

The average length of time that it took to clear the infections was around 7 days.

The results of the cultures in order of frequency was as follows:

1. Hemolytic Staphylococcus Aureus	18
2. Pseudomonas Aeroginosa	13
3. Bacillus Proteus	7
4. Hemolytic Staphylococcus Albus	6

Fungi and molds were found in 9 cases, and of these the Aspergillus Niger was identified in 6 cases. Diphtheroids were found in pure cultures in 2 cases and in another 15 cases in association with either Gram Positive or Gram Negative bacteria or Fungi. No correlation could be seen with the presence of diphtheroids in the cultures. Diphtheroids were found in a mixed culture in ten cases in association with Gram Positive bacteria, in 7 cases associated with Gram Negative, and in two cases of Aspergillus Niger.

Five of the patients treated in this group presented an acute infection of both ears and canals. It is interesting to note that in all 5 cases the bacteria cultured from both ears was the same. In three cases, the causative organism was the Hemolytic Staphylococcus Aureus, in one of which it was associated with the Candida Mold. In the other two cases the organism cultured was the Pseudomonas Aeroginosa.

Six patients, or 13%, developed an allergic reaction to the Furacin ear solution manifested by redness and swelling of the pre and post auricular areas with the formation of small discrete vesicles and profuse watering of the ear. It is interesting to point out that two of these 6 patients had pure cultures of diphtheroids.

As in the previous group the Furacin ear solution was found equally effective against the Gram Positive and Negative group of bacteria as well as against the Fungi.

It is noted that the incidence of infection by bacteria during the two seasons of the year is different.

In the first group studied in the summer months, the most frequent offender was the Pseudomonas Aeroginosa which was found in 14 cases or 35% as compared with the second group, studied in the winter months, where the most frequent offender was the Staphylococcus, found in 24 cases or 52%.

A total of 86 patients were studied in whom adequate clinical and bacteriologic follow up were available. A total of 12 patients or 14% developed untoward reactions, allergic in nature, which necessitated the discontinuance of the therapy. The remaining 74 cases responded well to the Furacin or Furacin Special Ear Solution therapy.

In comparing the results of cultures for the two groups it is apparent from this study that the Gram Negative bacteria tend to be more common in the warm weather months of the year when the humidity is higher.

SUMMARY AND CONCLUSIONS

A total of 86 patients with an acute infection of the external ear and canal were studied during two different seasons of the year, using two slightly different preparations. In the first group of 40 patients a Furacin Special Ear Solution was used. In the second group of 46 patients the regular Furacin ear solution was used. Cultures were done in every case for bacteria and fungi.

The two Furacin preparations were found equally effective against bacteria and fungi. Allergic reactions occurred in 14% of the cases.

ACKNOWLEDGMENT

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The laboratory work was done by Dr. A. Pomales Lebrón and Francisco Landrón Becerra, M.S. of the Landrón Medical Laboratory.

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EDITORIAL

¡ADELANTE CON EL ESCUDO AZUL!

Desde la década de 1930 hasta el presente se han venido desarrollando en los Estados Unidos de América, los planes de tipo Escudo Azul (seguro de servicios médico-quirúrgicos) y Cruz Azul (seguro de hospitalización). Estos son planes de seguro del tipo voluntario con fines no pecuniarios auspiciados por las asociaciones médicas estatales y locales con el beneplácito de la Asociación Médica Americana, como la solución más adecuada al creciente problema del alto costo de los servicios médicos modernos, salvaguardando a la vez la inviolabilidad de la relación voluntaria y directa de paciente y médico.

En Puerto Rico, se fundó en el año 1942, al amparo de la ley 152, la Cruz Azul de Puerto Rico que comenzó ofreciendo seguro de hospitalización al cual se le añadió en el 1946 seguro para servicios médico-quirúrgicos que se han designado de tipo Escudo Azul, pero que obviamente no ha sido, ni lo es, por no estar la Asociación Médica de Puerto Rico representada oficialmente en su Junta de Directores.

La Cruz Azul de Puerto Rico no ha llenado a cabalidad sus obligaciones con el público suscriptor, mayormente la clase media-obrera, al violar el precepto básico de libre elección de médico en sus Planes de Dispensario, amén de ganarse la animosidad y falta de cooperación de sus médicos asociados por la manera poco constructiva con que su administración se ha conducido en sus relaciones con ellos. Como consecuencia, su matrícula de suscriptores y médicos participantes ha permanecido en un estado de estancamiento numérico en los últimos 3-4 años, mientras las compañías de seguros y otros planes van aumentando su volumen.

La Asociación Médica de Puerto Rico, impelida por la creciente necesidad de ofrecerle a nuestra gran clase media-obrera y a su matrícula de médicos el tipo de Plan que verdaderamente pudiera solucionar el problema ya presentado, a la vez que obviara la necesidad de intervención gubernamental con fórmulas socializadas de alto costo y bajo rendimiento, tomó la iniciativa en forma dinámica y decidida propulsando los Proyectos de la Cámara 343, 344 y 345, en este momento ante la consideración de nuestra honorable Cámara de Representantes.

Compañeros, no basta con este esfuerzo de su Cámara de Delegados, Comités y Directiva, pues aún más importante es la aportación individual que cada uno de nosotros puede ofrecer hablándole o escribiéndole a sus representantes en las Cámaras Legislativas, a nuestro Gobernador a su debido tiempo, y ante todo,

firmando su endoso personal llenando los blancos que a ese fin les ha enviado la Presidencia a la brevedad posible a nuestra Asociación.

¡Adelante con el Escudo Azul de la Asociación Médica de Puerto Rico! Es deber ineludible y sagrado proveerle a nuestros pacientes servicios médico-quirúrgicos a precio razonable dentro de las normas de ética y buena práctica de nuestra profesión. Eso conseguiremos, apoyando ahora y en el futuro, nuestra Asociación y su Plan de Escudo Azul, aceptando si es necesario cualquier sacrificio económico por ver nuestro ideal realizado. Tenemos fe inquebrantable en el espíritu de abnegación y sacrificio que ha caracterizado la clase médica desde tiempos olvidados y no dudamos que ese mismo espíritu prevalece en nuestra matrícula. ¡Adelante con el Escudo Azul!

SECCION ADMINISTRATIVA

M E M O R A N D U M

A los : honorables señores legisladores
del : Dr. Luis R. Guzmán López, Presidente
Asociación Médica de Puerto Rico
Asunto : Proyectos 343, 344 y 345 para la creación de planes de seguro médico voluntario.

En el año 1930 tuvo su origen el primer plan de seguro médico voluntario en los Estados Unidos de América. Desde entonces estos planes se han ido expandiendo y multiplicando rápidamente, al extremo de que en la actualidad los mismos cubren en dicho país alrededor de 110 millones de habitantes, casi el 70% de la población total.

El desarrollo de estos planes es de tal magnitud que ha sido clasificado como uno de los más grandes movimientos colectivos en el mundo, ya que ni en el campo de la religión, ni en el de la política, ni en el de ningún otro campo sociológico se han podido agrupar 110 millones de personas para su propio beneficio en tan corto período de tiempo.

Estos planes han sido auspiciados por la Asociación Médica Americana y las asociaciones médicas de los diferentes estados de la Unión Americana, que han visto la necesidad imperativa de proveerle protección a la comunidad, haciéndole posible a muchos ciudadanos el acogerse voluntariamente a los beneficios máximos de servicios médico-quirúrgicos que ofrecen estos planes.

En Puerto Rico se comenzó a ofrecer el plan de Cruz Azul en el año 1942, siendo su objetivo principal brindar un seguro para servicios de hospitalización. Subsiguientemente se le añadieron al plan original servicios médico-quirúrgicos. Estos han sido de naturaleza limitada y nunca han satisfecho plenamente las necesidades y las ambiciones de los suscriptores. La clase médica del país tampoco ha estado satisfecha con las limitaciones en el funcionamiento de este plan y es por eso que desea brindar un tipo de servicio médico-quirúrgico comprensivo, abarcador y en consonancia con los principios bien establecidos que deben regir la organización de estos planes. Estos principios son:

1—Mantener la libre selección del médico por el suscriptor.

2—Conservar y respetar las relaciones personales entre paciente y médico.

3—La responsabilidad profesional debe ser asumida completamente por la profesión médica, que es la única que está cualificada para así hacerlo.

4—El plan debe tener la aprobación y el respaldo de la asociación médica local con el fin de mantener las normas más altas y estrictas en el rendimiento de estos servicios.

Este nuevo plan no sólo contempla la expansión de los beneficios profesionales para los suscriptores, sino que a su vez se compromete a hacerlo sin alterar las cuotas existentes. De esta forma puede expandirse considerablemente el número de ciudadanos que se beneficien del mismo.

En la actualidad solamente alrededor de 118,000 personas se benefician del plan de servicios médico-quirúrgicos que opera en nuestro país. Los objetivos de este nuevo tipo de plan son el de extender los beneficios médico-quirúrgicos a un grupo poblacional no menor de un millón de personas.

Este plan aliviarría grandemente la carga que actualmente pesa sobre nuestro gobierno en el rendimiento de servicios médico-quirúrgicos. Muchos de los enfermos que hoy día utilizan las facilidades gubernamentales podrían acogerse, sin gran sacrificio económico y con grandes ventajas, a estos planes de seguro médico voluntario.

Este nuevo plan contribuirá grandemente a resolverle el problema de los servicios médico-quirúrgicos a nuestra clase media que, por el progreso económico de nuestro país, es cada día más numerosa.

La Asociación Médica de Puerto Rico, consciente de la urgente necesidad de resolver este problema, respalda los proyectos 343, 344 y 345 de la honorable Cámara de Representantes, los que harán posible la instrumentación de planes de seguro médico-quirúrgico voluntario que llenen a cabalidad las necesidades ya descritas. Para lograr la feliz consecución de estos planes la clase médica está dispuesta a ir al sacrificio económico personal, si así lo requiriese el auspiciar estos planes.

En contraste con el número reducido de compañeros que respaldan al presente el tipo de servicio médico-quirúrgico que se ofrece dentro de la presente organización de La Cruz Azul, la casi totalidad de nuestros compañeros respaldarían y participarían con todo entusiasmo en planes que se estructuran de acuerdo con los proyectos anteriormente mencionados, como lo comprueba encuesta llevada a cabo recientemente por la Asociación Médica de Puerto Rico.

En resumen: ¿Por qué la Asociación Médica de Puerto Rico se ha tomado la iniciativa de respaldar proyectos de esta naturaleza? He aquí las razones:

1—El costo de los servicios médico-quirúrgicos modernos es tan alto que no le permite a la mayoría de los ciudadanos sufra-

garlos directamente sin sacrificios económicos. Un plan de esta índole ha probado ser la mejor solución a este problema.

2—Los planes de seguro médico-quirúrgico voluntario actualmente en vigor en Puerto Rico no brindan a los suscriptores la oportunidad de seleccionar libremente a su médico. El nuevo plan garantizará a los suscriptores esta libre selección.

3—La responsabilidad profesional de un plan de servicios médico-quirúrgicos debe recaer primordialmente sobre el médico y es éste el que precisamente debe velar por las normas más puras en la prestación de estos servicios.

4—En los planes actuales el público está recibiendo los beneficios de sólo una parte de la clase médica de Puerto Rico. Bajo el patrocinio de la Asociación Médica se conseguiría la participación, sino de la totalidad de la matrícula, por lo menos de su inmensa mayoría comprobado por la encuesta efectuada recientemente entre la matrícula activa de la Asociación Médica de Puerto Rico.

5—Aunque la remuneración que los médicos recibirán en planes de esta naturaleza resultará menor que la recibida en la actual práctica privada de su profesión, la clase médica ha demostrado su disposición a aceptar tal sacrificio como una aportación a la solución del alto costo de los servicios médico-quirúrgicos modernos.

6—Este nuevo tipo de plan ofrecerá al suscriptor una protección contra riesgos de enfermedad o accidentes mucho más amplia y comprensiva que la que se le ha venido ofreciendo hasta la fecha.

7—Nuestra creciente clase media tendrá en estos planes un medio efectivo para enfrentarse a sus necesidades médico-quirúrgicas.

Los proyectos 343, 344 y 345 se agrupan por tener una idea básica en común, a saber: autorizar la creación en Puerto Rico del Escudo Azul y de otras asociaciones similares con fines no pecuniarios para prestar servicios médico-quirúrgicos exclusivamente, limitando a La Cruz Azul de Puerto Rico y a otras asociaciones similares que se pudieran organizar en el futuro al amparo de la Ley 152 del 1942, a prestar única y exclusivamente servicios de hospitalización. Se faculta además, al Comisionado de Seguros a contratar con asociaciones de tipo Escudo Azul los servicios médico-quirúrgicos a ser ofrecidos a los funcionarios y empleados del gobierno de Puerto Rico y a contratar con asociaciones de tipo Cruz Azul los servicios hospitalarios para estas mismas personas.

P. de la C. 345: Mediante este proyecto se conseguiría legislación de carácter general para que se puedan organizar asociaciones con fines no pecuniarios para rendir exclusivamente servicios médico-quirúrgicos. Esa operación estará bajo la supervisión del Comisionado de Seguros. No se afectarán los servicios que puedan prestarse con arreglo a la Ley de Compensaciones por Accidentes

del Trabajo, por la Asociación de Maestros de Puerto Rico, por la Sociedad Española de Auxilio Mutuo y Beneficencia o por las cooperativas que se pudieran organizar en el futuro.

Los principios básicos de este proyecto pueden enumerarse en la siguiente forma:

I. Permite la creación de asociaciones con fines no pecuniarios, cuyo objetivo principal es el rendir servicios a sus asociados.

II. La Junta de Directores estará compuesta de ocho médicos en el ejercicio legal de la medicina y cirugía, seleccionados por la Asociación Médica, y de ocho personas que representen el interés público y que fueren suscriptores del plan, que serán seleccionadas por los suscriptores del plan en asamblea anual.

III. Todo contrato extendido por dicha asociación a cualquier suscriptor constituirá una obligación directa del médico o médicos con los cuales haya contratado la asociación para tales servicios.

IV. Todo contrato con el suscriptor podrá ser individual, por familia o por grupo.

V. Las cuotas que hubieran de pagar los suscriptores o socios por servicios médico-quirúrgicos estarán sujetas a la previa aprobación del Comisionado de Seguros, quien determinará, a base de estudios actuariales, si las mismas son adecuadas para cubrir los gastos administrativos y el pago de honorarios.

VI. Los honorarios a pagar por la asociación a los médicos-cirujanos que presten servicios médico-quirúrgicos a los suscriptores, serán determinados por la Junta de Directores previo asesoramiento por un comité de médicos seleccionados por dicha Junta, y no entrarán en vigor hasta no ser aprobados previamente por el Comisionado de Seguros.

VII. Ninguna asociación sujeta a las disposiciones de esta Ley podrá celebrar contratos sin estar previamente autorizada por el Comisionado de Seguros mediante el certificado correspondiente.

VIII. En ningún año la asociación habrá de emplear más de un 20% del ingreso por concepto de cuotas de suscriptores para gastos de administración.

IX. El Comisionado de Seguros practicará o hará que se practique, por lo menos cada dos años, un examen de las condiciones de la asociación.

X. El presupuesto general estará sujeto a la aprobación previa del Comisionado de Seguros.

XI. Se creará una reserva para gastos extraordinarios ocasionados por una epidemia o catástrofe o por cualquier otro acontecimiento.

XII. El Comisionado de Seguros tendrá autoridad para aprobar reglamentos para la debida ejecución de la ley.

XIII. Toda asociación organizada al amparo de la ley:

(a) Podrá contratar con el gobierno, sus agencias e instrumentalidades y con los gobiernos municipales, con el fin de prestar servicios médico-quirúrgicos a sus funcionarios, empleados y dependientes de estos, y a personas necesitadas o a cualquiera otra persona, y asimismo podrá recibir o aceptar de dichas agencias gubernamentales pagos que cubran la totalidad o parte del costo de la suscripción para proveer dichos servicios.

(b) Podrá recibir y aceptar de agencias privadas, corporaciones, asociaciones, uniones obreras o grupos, pagos que cubran la totalidad o parte del costo de prestar servicios médico-quirúrgicos a sus miembros, funcionarios y empleados y cualesquiera otras personas.

(c) No podrá suministrar servicios médico-quirúrgicos sino a través de médicos-cirujanos debidamente licenciados para ejercer su profesión en el Estado Libre Asociado.

(d) No podrá intervenir en las relaciones entre el médico y su paciente, entendiéndose que el suscriptor tendrá la libre elección de médico en todo momento.

(e) No podrá imponer restricción alguna a los médicos que den tratamiento a los suscriptores en cuanto a los métodos de diagnóstico o tratamiento que ellos empleen.

P. de la C. 344: Este proyecto enmienda las secciones 1, 3, 6 y 9 de la Ley 152 del 9 de mayo de 1942. Se elimina la autoridad para rendir servicios médico-quirúrgicos y servicios de dispensario médico que tienen las Asociaciones de tipo Cruz Azul y se prohibe a estas asociaciones que se dediquen a prestar servicios médico-quirúrgicos o de dispensario. Esas agrupaciones estarán limitadas a prestar servicios **hospitalarios.**

Las enmiendas a la sección 3 de Ley 152 del 1942 cambian la naturaleza de la Junta de Regentes de tal tipo de asociación en consonancia con el hecho de que solamente prestarán estas asocia-

ciones servicios hospitalarios. Esto implica que en la Junta de Directores estarán representados solamente los suscriptores y los hospitales, en igual número.

Las enmiendas a la sección 3 de la Ley 152 del 1942 cambian la 152 tienen por objeto hacer uniformes dichas secciones con lo que disponen iguales secciones en el P. de la C. 345. Se especifica que el presupuesto anual de gastos de una asociación dedicada a rendir servicios hospitalarios estará sujeto a la aprobación del Comisionado de Seguros y que dicho presupuesto no podrá ser enmendado sin la previa aprobación del Comisionado. Este proyecto tendrá vigencia un año después de su aprobación, de manera que se le da tiempo para poder organizarse a una asociación que vaya a prestar servicios médico-quirúrgicos antes de que entren en vigor las disposiciones del proyecto que prohibe a la Cruz Azul y a otras asociaciones similares el prestar dichos servicios médico-quirúrgicos.

P. de la C. 343: Este proyecto autoriza al Comisionado de Seguros a contratar los servicios médico-quirúrgicos para los empleados y funcionarios del gobierno estatal con una asociación que se organice al amparo del P. de la C. 345, y los servicios hospitalarios para estos mismos empleados y funcionarios con cualquier asociación organizada o que en el futuro se organice bajo las disposiciones de la Ley 152 del 1942.

Respetuosamente,

Luis R. Guzmán López, M.D.
Presidente

SECCION ADMINISTRATIVA

CARTA MENSUAL DEL PRESIDENTE

Primera Reunión Ordinaria de la Cámara de Delegados: El sábado, 12 de abril, a las 3:00 de la tarde, se llevará a efecto en el domicilio de nuestra Asociación la primera reunión ordinaria de la Cámara de Delegados, bajo la presidencia del doctor Enrique Pérez Santiago.

En dicha reunión ordinaria se tratarán asuntos de verdadero interés para nuestra agrupación, entre otros los siguientes:

(a) Considerar nombramiento de un Director Ejecutivo, lo cual conlleva el decretar un aumento razonable en la cuota;

(b) Consideración de una proposición para un seguro de vida colectivo mediante el pago de una tarifa sumamente razonable;

(c) Status de la legislación médica (Escudo Azul, médicos extranjeros, etc.)

Suplicamos a los compañeros delegados, tanto a los propietarios como a los suplentes, hagan los arreglos pertinentes para asistir a esta primera reunión de la Cámara y darnos su concurso para el mejor éxito de nuestras deliberaciones. Anote esta fecha en su calendario de actividades médicas: SABADO, 12 de abril, a las 3:00 de la tarde.

❖ ❖ ❖

Nuevos Delegados: Nos complacemos en transcribir a continuación los nombres de los compañeros que actuarán como delegados por los Distritos Este y Sur, según certificación que obra en la Secretaría de nuestra Asociación:

Distrito Este

Antonio Rullán
Víctor M. Rivera
A. Barreto Domínguez
Ramón A. Sifre, Jr.
R. Cuevas Zamora
Carlos Guzmán Acosta
J. Basora Defilló
Gualberto Rabell
José Berio
José A. Peña
Agustín M. de Andino
Bernabé Lima Báez
José S. Licha
Héctor Feliciano
F. Hernández Morales
Héctor M. Sampayo
Pedro J. Collazo
Pablo Luis Morales
Angel M. Mattos
Fernando Vallecillo
José García García
Jaime F. Pou
Francisco Berio Suárez
Egidio S. Colón Rivera
Víctor Marcial

Suplentes

Eladio Montalvo Durand
Oscar Costa Mandry
Ricardo F. Fernández
Rafael A. Gil
Anthony Lombardi

Distrito Sur

Luis F. Sala
Carlos F. Jiménez Torres
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A .García Soltero
Enrique A. Vicéns
A. Fernández Durán
Héctor F. Rodríguez
José del Prado
Armando Antommattei

Suplentes

Orlando Salichs
Julio C. Roca
Francisco Barnés
Luis A. Rosario
Tito Mattei

❖ ❖ ❖

El Médico Como Optómetra: Mediante una supuestamente tentadora oferta y diseminando información errónea en el sentido de que la Asociación Médica de Puerto Rico no tiene objeción a que los médicos desempeñen las funciones de optómetras, una conocida firma comercial dedicada al negocio de optometría está haciendo esfuerzos por contratar los servicios de médicos para atender sus negocios en distintos pueblos de la Isla.

Una vez más llamamos la atención de los compañeros asociados hacia el hecho de que ocupar un cargo de tal naturaleza está reñido con los Cánones de Etica de la profesión médica. La Cámara de Delegados de nuestra Asociación así lo ratificó en su reunión ordinaria de noviembre ppdo. y esperamos que los compañeros asociados se abstengan de entrar en esta clase de negocios.

Los médicos NO asociados que se han unido a dicha firma están fuera de la jurisdicción de nuestra Asociación; pero sus nombres aparecen en nuestros records como violadores de la ética profesional y como tales serán tratados en el futuro.

❖ ❖ ❖

Actividades de Nuestras Secciones: Transcribimos a continuación, para conocimiento general, información de interés en relación con algunas de las secciones de nuestra Asociación.

Sección de Pediatría

La nueva directiva de la Sección de Pediatría está integrada como sigue:

Presidenta: Dra. Dolores I. Méndez Cashion
Pres. electo: Dr. Osvaldo González
Secretaria: Dra. Carmen L. Berio
Tesorero: Dr. Juan F. Jiménez

La sección auspiciará los siguientes actos, para los cuales se ha extendido invitación a toda la matrícula de la Asociación:

Conferencias a cargo de la doctora Edna H. Sobel del Albert Einstein College de Medicina.

1. MIERCOLES, 9 de abril, 8:00 a 9:00 de la mañana - Anfiteatro de la Escuela de Medicina en el Hospital de la Capital:
   Tema: ENDOCRINE DISTURBANCES ON GROWTH: Presentation and Discussion of Cases.
2. JUEVES, 10 de abril, 8:30 de la noche — Edificio de la Asociación Médica.
   Tema: (Será anunciado oportunamente).

Sección de Cirugía General

La nueva directiva de la Sección de Cirugía General de nuestra Asociación está integrada por los siguientes compañeros:

Presidente: Dr. E. M. de Hostos
Vicepresidente: Dr. Benigno González
Secretario: Dr. Ramón Isales
Tesorero: Dr. R. Mejía Ruíz
Vocales: Dr. A. S. Casanova Díaz
Dr. Luis F. Sala
Dr. Manuel A. Astor
Dr. Nelson Perea

La directiva tomará posesión el JUEVES, 10 de abril, a las 7:30 de la noche, y con tal motivo se celebrará un coctel-buffet en honor a los miembros de la Sección y sus esposas.

Seccion de Medicina General

La Sección de Medicina General, que preside el doctor Fernando Vallecillo, celebrará una reunión de carácter social en el Casino de Puerto Rico el SABADO, 12 de abril, a las 7:30 de la noche. El propósito de dicha reunión es promover un mayor acercamiento entre el grupo de médicos generalistas y sus respectivas esposas, y es de esperar que a la misma concurra un nutrido grupo.

Esta Sección ha venido tomando gran auge durante los últimos meses. Para actividades futuras solamente serán invitados aquellos compañeros que hayan llenado el impreso de solicitud que ha circulado entre los médicos generalistas. Si usted se dedica a la práctica general de la medicina e interesa formar parte de esta Sección sírvase llenar y devolver el impreso que le fué remitido. Si se le ha extraviado o no lo ha recibido puede solicitar un nuevo impreso al doctor Fernando Vallecillo.

❖ ❖ ❖

Asamblea Anual - Noviembre 18-22, 1958: El doctor Calixto A. Romero, presidente del Comité Científico de nuestra Asociación, nos ha remitido la siguiente nota:

El Comité Científico desea comunicar a todos los miembros de la Asociación que deseen presentar un trabajo en la próxima asamblea anual que deberán someter el título del mismo para el día 31 de mayo, con el fin de preparar la programación. Un resumen del trabajo a ser presentado deberá ser sometido a más tardar el día 30 de septiembre, de manera que el comité pueda organizar el orden de la presentación de las contribuciones científicas.

❖ ❖ ❖

Relaciones Entre Médicos y Hospitales: El doctor Carlos Guzmán Acosta, presidente del Comité de Relaciones entre Médicos y Hospitales, desea suplicar a la matrícula, por nuestro conducto, se sirvan referir a su comité aquellos problemas que consideren deban ser tratados por el Comité de Relaciones Entre Médicos y Hospitales.

❖ ❖ ❖

Solicitudes de Ingreso por Considerar: El Comité de Credenciales, que preside el doctor A. Otero López, tiene ante su considención las solicitudes de ingreso de los siguientes compañeros:

Miembros Regulares:

1. Dr. Carlos R. Macossay Negrín, de Río Piedras
2. Dr. Francisco Febles Vizcarrondo, de San Juan
3. Dr. Manuel A. de Jesús González, de Arecibo
4. Dr. Emilio F. Trilla Piñero, de Carolina
5. Dr. Gabriel Zamorano Estapé, de Ponce

Miembros Afiliados:

1. Dr. José Enrique Figueroa Ballester, de Santurce
2. Dr. Miguel Angel Quetglas, Jr., de Río Piedras

Si usted tiene alguna información de interés relacionada con alguno de estos candidatos, le suplicamos tenga la bondad de hacerla llegar al comité por conducto de su presidente.

❖ ❖ ❖

Vacante en el Hospital Municipal de Arecibo: Nos comunica el buen amigo Darío Goitía, Alcalde de la Villa del Capitán Correa, que hay una plaza de médico municipal vacante en Arecibo, con sueldo de $500.00 mensuales. Los compañeros interesados pueden comunicarse directamente con el señor Alcalde Goitía, o con el doctor Johnny López, Director Médico del Hospital Municipal de Arecibo.

Luis R. Guzmán-López, M.D.
Presidente

Asociación Médica de Puerto Rico

ASAMBLEA ANUAL

Noviembre 18-22, 1958

ASAMBLEA ANUAL

ASOCIACION MEDICA DE PUERTO RICO

Noviembre 19-23, 1957.

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DE LA

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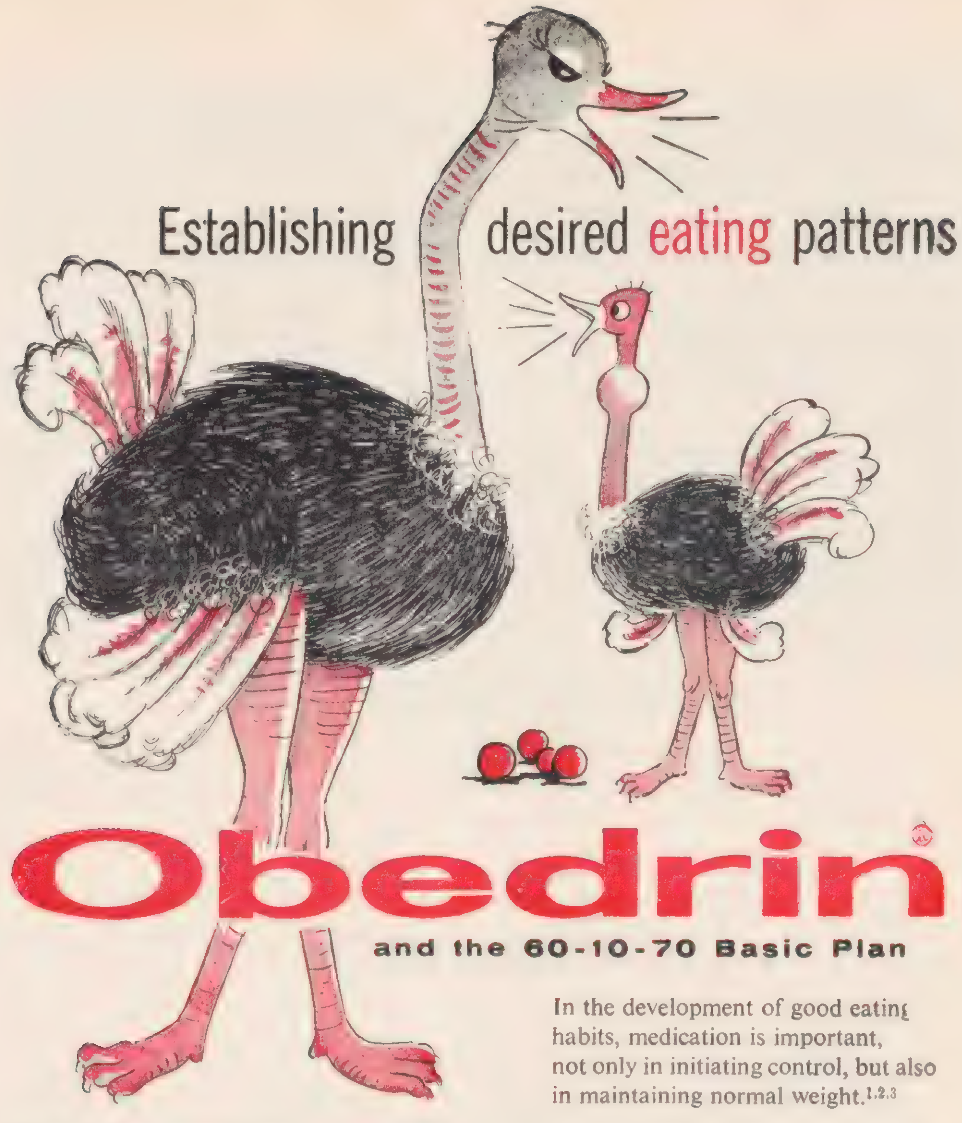
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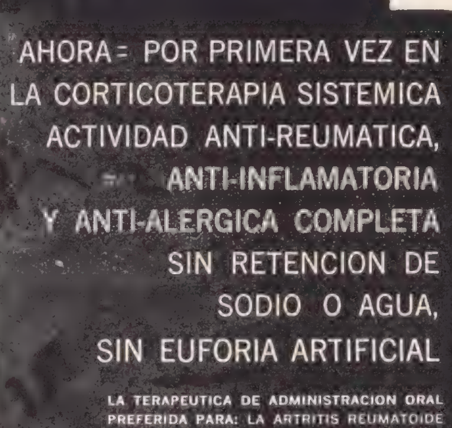
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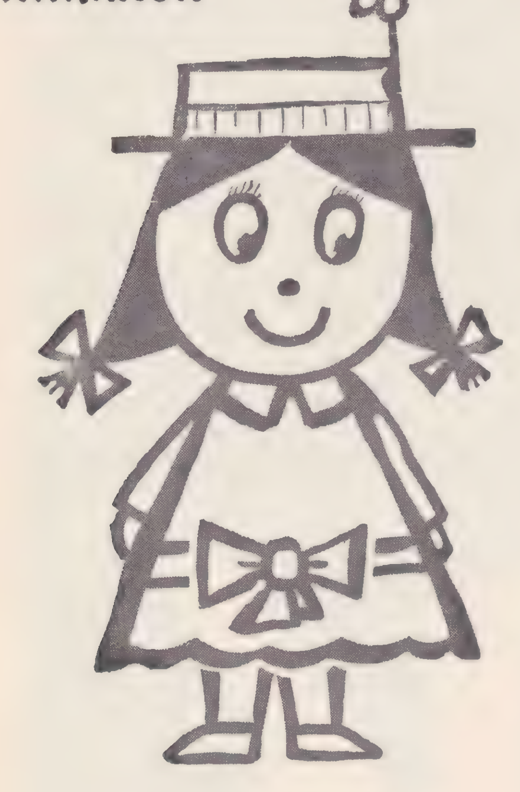

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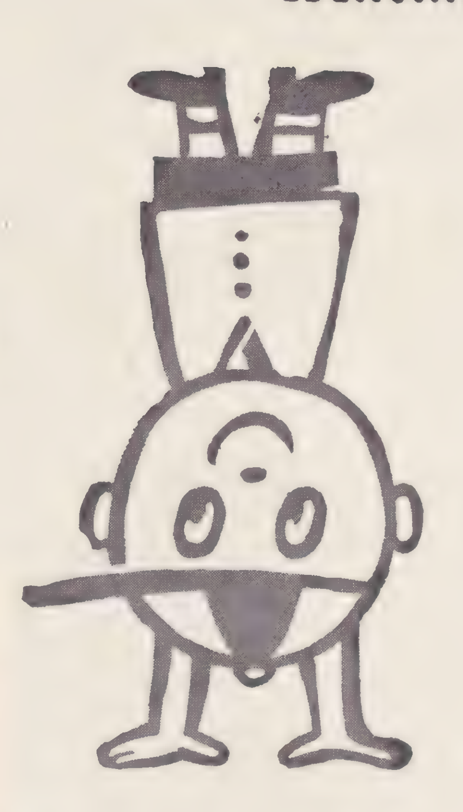

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DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 50 ABRIL, 1958 NO. 4

EVALUATION OF THE TESTS FOR SERUM TRANSAMINASE ACTIVITY, AND ETHER-EXTRACTABLE BILIRUBIN IN URINE IN THE JAUNDICED PATIENT

J. M. BERIO, M.D., F. HERNÁNDEZ-MORALES, M.D.,** R. RAMIREZ-WEISER, M.D.,* MERCEDES V. DE TORREGROSA, PH.D.,*** and NILDA M. TORRES, B.S., M.T.**

INTRODUCTION

In the differentiation of the various types of jaundice several combinations of the tests of liver function have been employed with variable success and lack of general acceptance. It is recognized that no one test can be considered as a reliable index of liver function under all conditions. It is also well accepted that no test or combination of tests of liver function is a substitute for a careful clinical appraisal of the jaundiced patient.[1] The study of the formation, metabolism and excretions of bilirubin offers critical information for the interpretation of the various mechanisms leading to the development of jaundice.[11,12,1] In the jaundiced patient with bilirubinuria, the urinary bilirubin has been found to yield almost exclusively bilirubin conjugated with glucuronic acid, which is direct reacting bilirubin.[4] Tests which demonstrate diffuse parenchymal liver damage, early in the course of liver disease are also of great value in the differential diagnosis of jaundice. An example of these is the elevation in the serum transaminase activity (SGO-T and SGP-T) which has been considered of diagnostic importance in the determination of liver cell injury.[7,13] In this paper we will present the results obtained in 26 jaundiced patients by using the tests for serum transaminase activity[8] and ether-extractable bilirubin in urine,[5] besides the serum bilirubin partition.[10] The material is representative of the two types of jaundice presenting the greatest difficulties for their differentiation, namely, hepatogenous and extra-hepatic obstructive jaundice.

* From Bayamón District Hospital.
** From Auxilio Mutuo Hospital.
*** From the San Juan City Hospital.

METHODS

The colorimetric method for the estimation of serum transaminase activity (SGO-T and SGP-T) was employed.[8] This method is simple and requires no enzymes as reagents, but is somewhat less accurate than the other methods (spectrophotometric and chromatographic.)[6]

Normal values determined in 14 blood donors are as follows:

SGO-T = (15-41) units
SGP-T = (10-45) units

The serum transaminase activity was considered normal from 0.50 units, slightly elevated from 51-150 units, moderately elevated from 151-300 units, and markedly elevated if above 300 units.

The test for ether-extractable bilirubin in urine (EEB-U) as described by Wainrach was modified.[5] Vigorous shaking of the urine-ether mixture was eliminated because it may result in the formation of a "gel". It is recommended that mixing be carried out by gentle inversion of the test tube about 50 times. This is followed by centrifuging at slow speed (1500 r.p.m.) for 3 minutes to obtain a clear supernatant. The reading of the % transmission of light in the supernatant-ether-layer is taken in the Coleman Jr. Spectrophotometer at 420 mu, using ether as the control blank.

Normal values for EEB-U in 10 healthy individuals ranged from 65 to 94% of light transmission. In 10 patients without suspected liver disease or jaundice similar values were obtained, ranging from 65 to 95%.

The ether-extractable bilirubin in urine test was graded as definitely positive (+) if the percentage of light transmission was below 50; doubtful (±) if the percentage of light transmission was 51-60; and negative if the percentage of light transmission was above 60.

In four cases of extrahepatic obstructive jaundice having a positive EEB-U test and employing the method of Golden & Snavely, the bilirubin extracted in the supernatant ether layer ranged between 5.4 to 34% of the total bilirubin in the urine with an average of 19%.[9]

The serum bilirubin was determined by the method of Malloy and Evelyn.

ANALYSIS OF MATERIAL

Twenty six cases of jaundice were adequately studied. These are divided into three groups: I) extrahepatic biliary obstructive jaundice; II) hepatogenous jaundice due to acute, diffuse liver injury; III) hepatogenous jaundice due to chronic liver disease.

Group I includes 9 patients, 5 males and 4 males, ages ranging from 58 to 92 years. In 9 cases the diagnosis was confirmed by laparotomy and in 5 of them by histologic examination also. Two patients had cancer of the ampulla of Vater, six had cancer of the pancreas, one had cancer of the gall bladder. The SGO-T, or SGP-T activity or both were slightly to moderately elevated in all cases except one case who had a marked elevation in SGO-T once. The ether-extractable bilirubin in urine was definitely positive in seven cases, doubtful in two cases.

TABLE I

EXTRA-HEPATIC BILIARY OBSTRUCTIVE JAUNDICE

Case No.	Sex	Age	Diagnosis	Serum Transaminase		Ether Extractable Bilirubin in urine
				SGO	SGP	
(1) RRM 141860	M	58	Cancer Amp. Vater	sl elev	sl elev	+
(2) PL 141840	M	58	" Pancreas	mod elev	mod elev	+
(3) AGC 141654	F	92	" Pancreas	sl elev	sl elev	+
(4) MC 140270	F	58	" Pancreas	mod elev	--	±
(5) AL 142630	F	66	" Gall Bladder	mod elev	mod elev	±
(6) JR 137952	M	56	" Pancreas	sl elev	--	±
(7) AB 120201	F	70	" Pancreas	mod elev	--	+
(8) PV	M	60	" Amp. Vater	sl elev	normal	+
(9) EM 143770	M	75	" Pancreas	marked elev	mod elev	+

In Group II, hepatogenous jaundice due to acute liver injury, a total of 12 cases was included, 5 females and 7 males, ages ranging from 11 to 87 years, with ten patients below the age of 40 yrs. In eleven the diagnosis was acute hepatitis (most likely of viral etiology), and one case had pericholangitic hepatitis with associated cholelithiasis. In the latter case the diagnosis was confirmed by biopsy of the liver, and operative cholangiography showed a normal extrahepatic biliary system. This case had a slight elevation of the SGO-T and SGP-T activity, and a definitely positive test for ether-extractable bilirubin in urine. In 10 of the 11 cases of acute hepatitis who had determinations of SGP-T there was a

marked elevation; while the SGO-T was markedly elevated in ten and moderately elevated in one case. That is, all the cases of acute hepatitis except that with pericholangitic hepatitis had a marked elevation of SGO-T or SGP-T or both. The EEB-U was definitely positive in 4 cases, doubtful in 2 cases and negative in 6 cases.

TABLE II

HEPATOGENOUS JAUNDICE, ACUTE LIVER INJURY

Case No.	Sex	Age	Diagnosis	Serum Transaminase Activity SGO	SGP	Ether Extractable Bilirubin in urine
(1) ICL 111255	F	70	Pericholangitic Hepatitis Cholelithiasis	sl elev	sl elev	+
(2) HM 141066	M	23	Acute Hepatitis	mod elev	marked elev	—
(3) JEC 150170	M	16	Acute Hepatitis	marked elev	marked elev	+
(4) JOR 140756	M	52	" "	marked elev	--	—
(5) FSR 141699	M	22	" "	marked elev	marked elev	—
(6) NR 142082	F	40	" "	marked elev	marked elev	+
(7) JM 142221	M	13	" "	marked elev	marked elev	—
(8) EJ 80919	F	11	" "	marked elev	marked elev	—
(9) ELQ 150102	F	12	" "	marked elev	marked elev	—
(10) AM 142649	M	28	" "	marked elev	marked elev	±
(11) TT 116982	M	32	" "	marked elev	marked elev	+
(12) LA	F	87	" "	marked elev	marked elev	±

Group III, hepatogenous jaundice secondary to chronic liver disease and fatty liver, included five cases, 3 females and 2 males, ages ranging from 26 to 55 yrs. The diagnosis in these cases was based on clinical findings, histologic examination was not performed. The SGO-T was markedly elevated while the SGPT-was moderately elevated in one case. One case had normal SGP-T and SGO-T, while three cases showed a slightly elevated SGO-T and a normal SGP-T. The EEB-U was negative in 3 cases, doubtful in one case, and definitely positive in another case.

TABLE III

HEPATOGENOUS JAUNDICE, CHRONIC LIVER DISEASE

Case No.	Sex	Age	Diagnosis	Serum Transaminase Activity		Ether Extractable Bilirubin in urine
				SGO	SGP	
(1) CGM 141301	F	26	Cirrhosis liver (active) Schistosomiasis Bleeding varices, esophageal	marked elev	mod elev	—
(2) FR 141373	F	38	Cirrhosis liver (active)	sl elev	normal	—
(3) JFS 126184	M	43	Cirrhosis liver (active) Schistosomiasis	sl elev	normal	+
(4) BC	M	66	Cirrhosis liver (active)	normal	normal	±
(5) GCA	F	38	Fatty liver (Cirrhosis?)	sl elev	normal	—

Serial determination of serum transaminase activity and ether-extractable bilirubin in urine were performed in 6 cases of extra-hepatic biliary obstruction. Figure I presents the pattern for EEB-U that appears to show all possible results in this condition. The percentage of light transmission gradually diminishes to a point below 40 and then rises to normal values. Figure II shows a section of the liver in this case in the area of the central vein revealing bile staining of the cystoplasm of hepatic cells, and plugging of biliary canaliculi. Parenchymal liver damage is insignicant. Figure III shows the descending limb of the curve, while figure IV shows the ascending limp. Figure V is a section of the liver in the case shown in figure III. It shows prominent plugging of biliary canaliculi, and bile staining of the cytoplasm of the hepatic cells. There is some evidence of hepatic cell injury with a finely vacuolated cytoplasm. An occasional inflammatory cell is seen in the sinusoids. Figure VI is a section of the liver in the case shown in figure IV. It reveals bile plugging of canaliculi and slight staining of the cytoplasm of the hepatic cells. The sinusoids are dilated and there is minimal evidence of hepatic damage. There were three cases similar to Figure III and two cases like Figure IV. It is observed in these figures that the direct hyper-bilirubinemia is sustained, while the serum transaminase activity varies from a slight to moderate elevation.

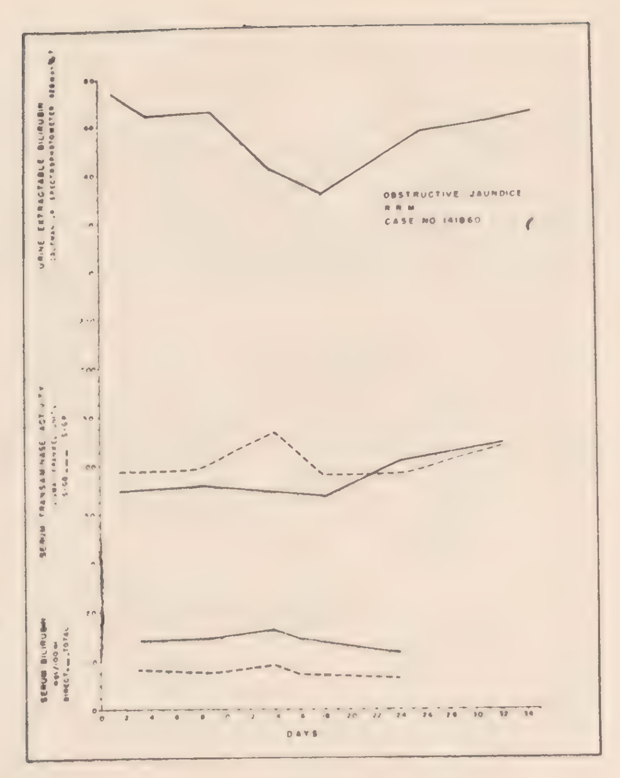

URINE EXTRACTABLE BILIRUBIN
OBSTRUCTIVE JAUNDICE
R R M
CASE NO 141860
SERUM BILIRUBIN
DAYS

FIG. I

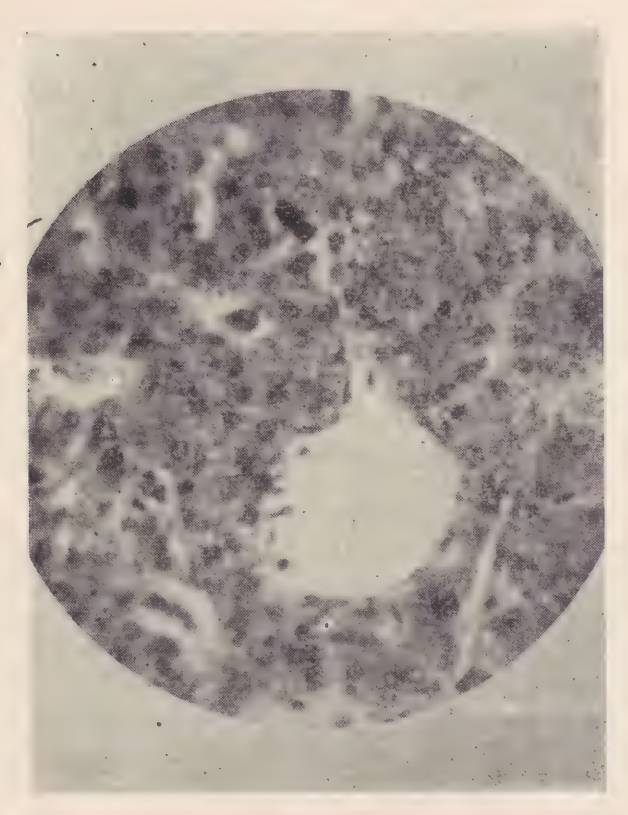


FIG. II

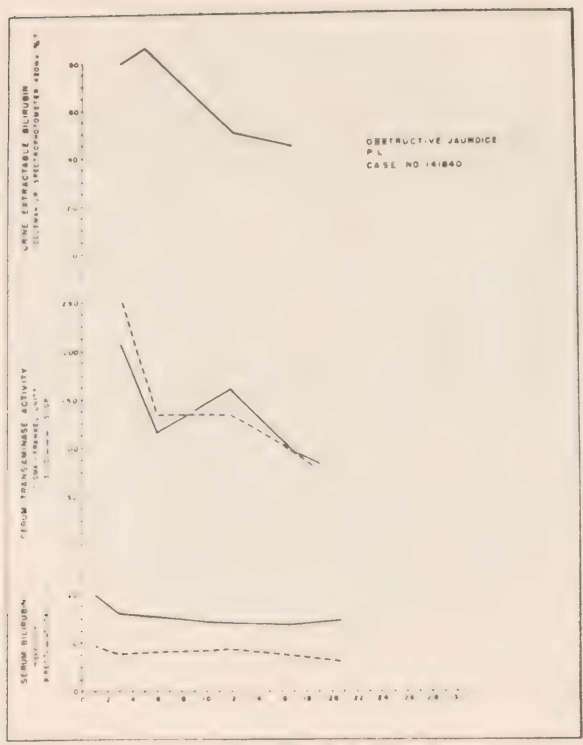


FIG. III

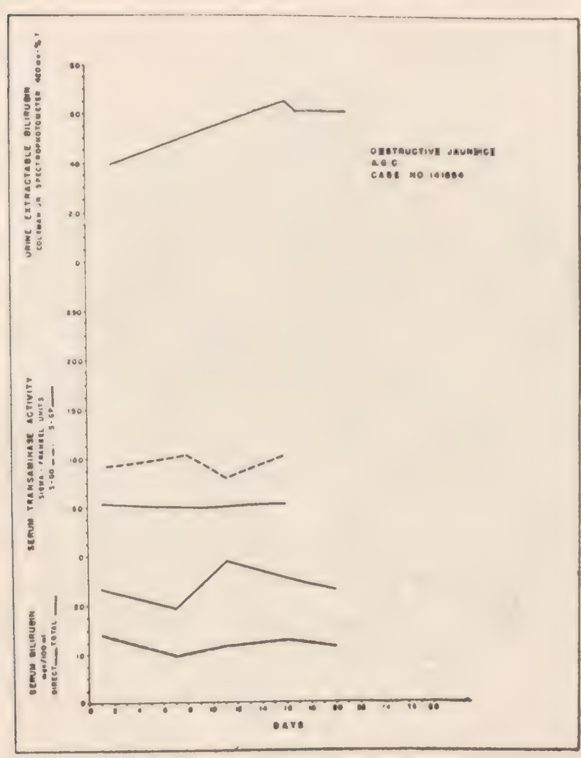


FIG. IV

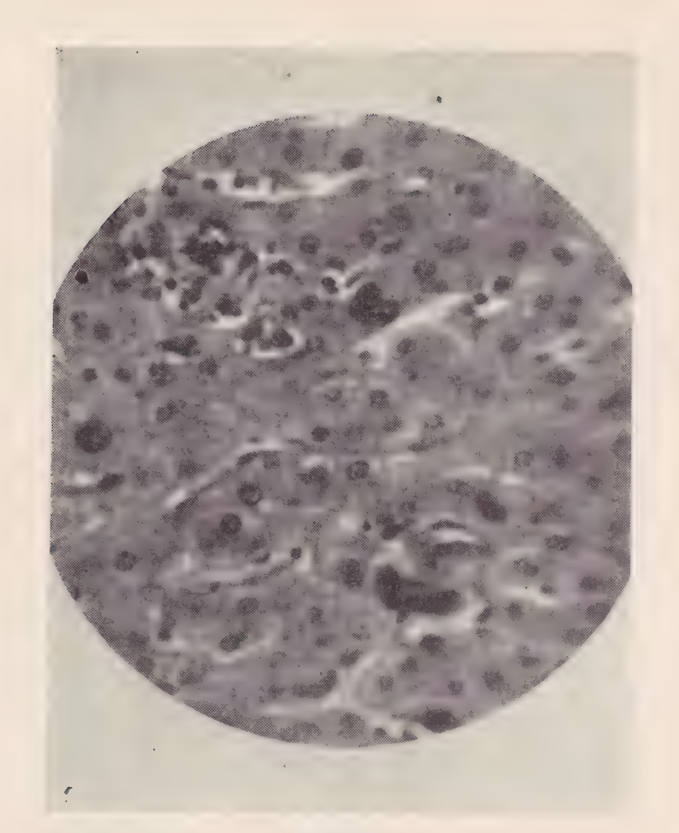


FIG. V

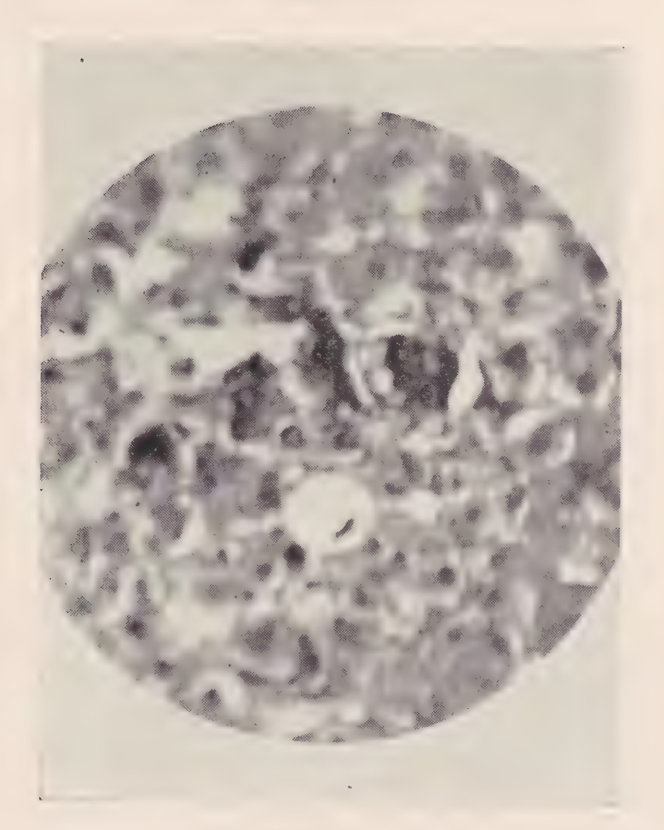


FIG. VI

The case of pericholangitic hepatitis (intrahepatic biliary obstruction) showed a sustained hyper bilirubinemia, a slightly elevated serum transaminase activity (border-line) and a positive EEB-U followed by an early rise to doubtful values. (Figure VII). Figures VIII and IX are sections of the liver in this case. The low power view shows evidence of plugging of biliary canaliculi, bile staining of the cytoplasm of hepatic cells, and prominent hyperchromatism of hepatic cell nuclei, with numerous large nuclei. A high power view shows the nuclear alterations (double nuclei, large nuclei) indicative of hepatic cell regeneration, and small foci of inflammatory cells predominanty lymphocytes and plasma cells with an occasional eosinophile and polymorphonuclear leukocyte.

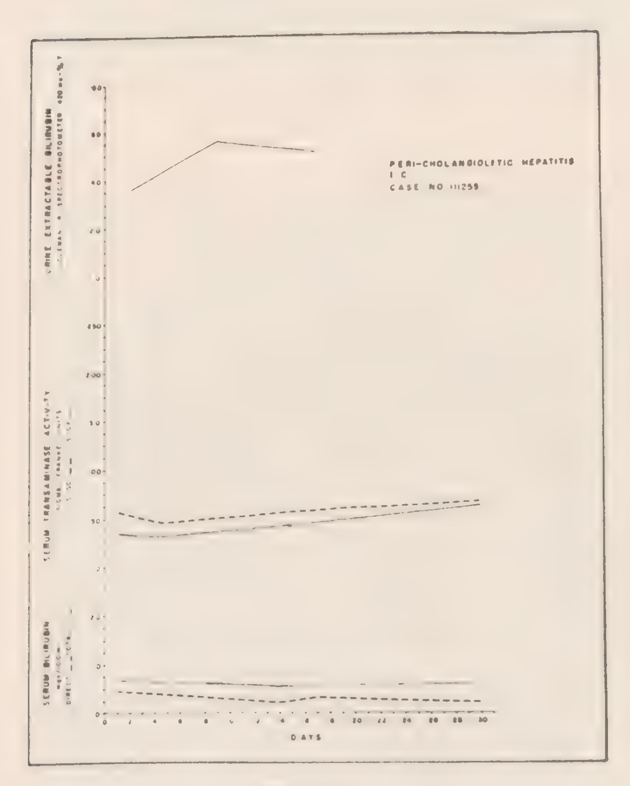


FIG. VII



FIG. VIII

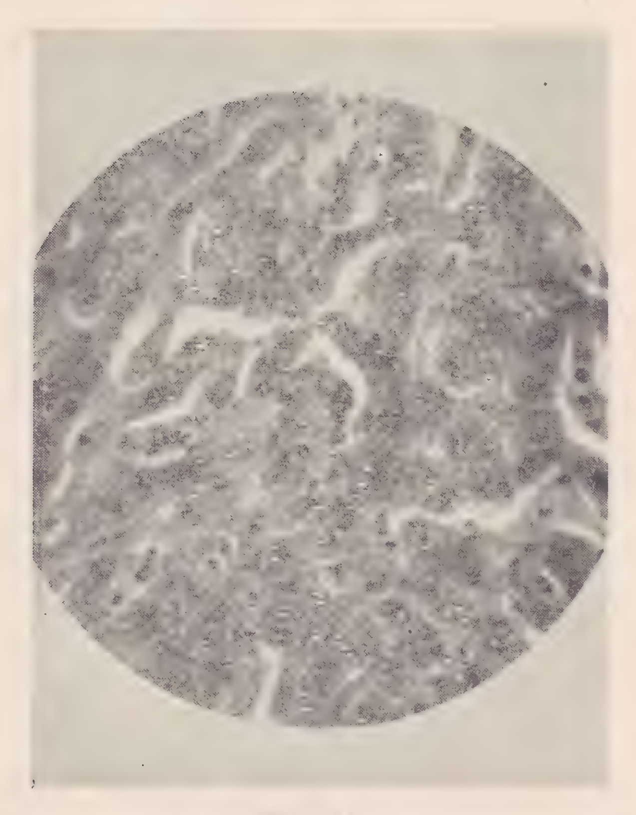


FIG. IX

The cases of acute hepatitis followed two patterns. Five cases showed a doubtful or definitely positive EEB-U followed by a rapid rise to normal values. This was accompanied by a corresponding drop in serum transaminase activity and serum bilirubin. (Figure X). In the remaining six cases the EEB-U was persistently normal, while the serum bilirubin and serum transaminase activity dropped as in the previous 5 cases. (Figure XI).

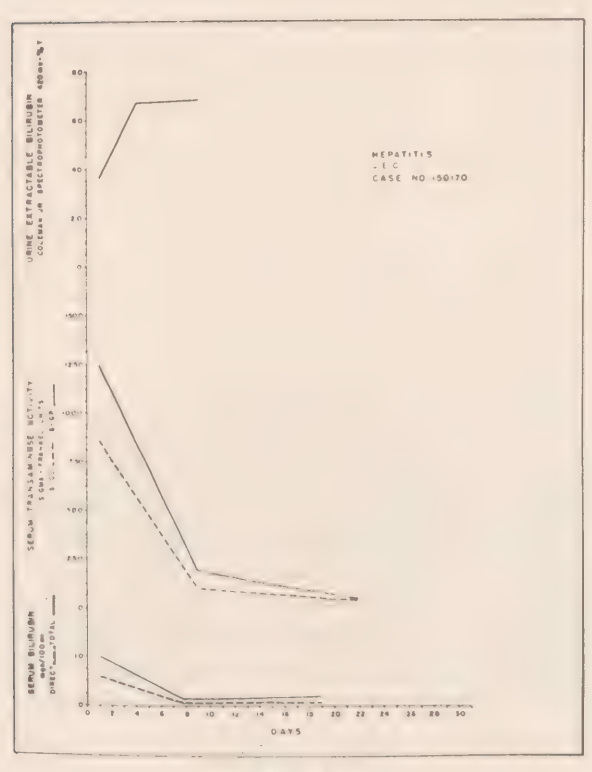


FIG. X

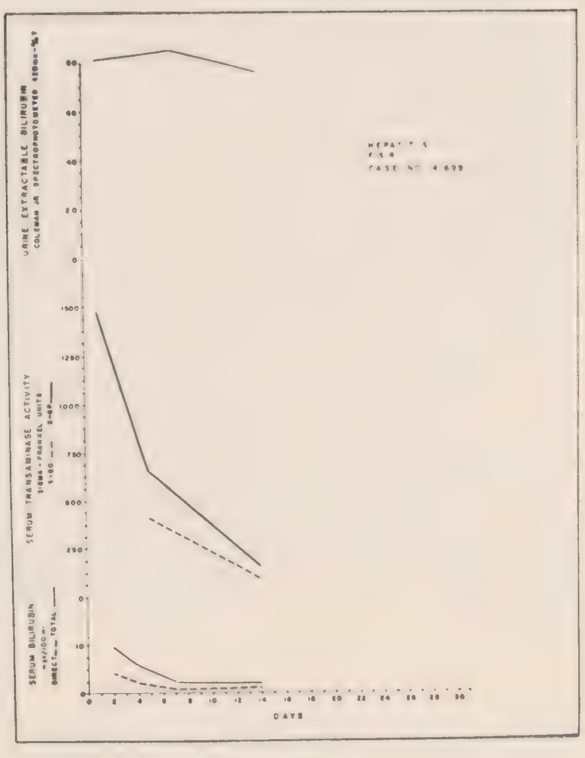


FIG. XI

Serial tests in the cases of jaundice secondary to cirrhosis of the liver and fatty liver showed a similar pattern in three of them. The EEB-U was normal. The SGO-T and SGP-T were normal or slightly elevated, except in one case where there was a marked elevation of SGO-T and moderate elevation of SGP-T. The serum bilirubin subsided gradually to almost normal levels. One case had a positive EEB-U on one occasion, a slightly elevated SGO-T, a normal SGP-T, and a gradual drop in serum bilirubin. The other case showed a doubtful EEB-U on one occasion, normal SGO-T and SGP-T and gradual disappearance of hyperbilirubinemia. (Fig. XII)

DISCUSSION

From the data presented it appears that serial determinations of serum transaminase activity and ether-extractable bilirubin in urine are of diagnostic value in the differentiation of non-obstructive from obstructive jaundice.

A positive EEB-U test in a jaundiced patient is indicative of an obstructive mechanism. The obstruction may be intra or extra-hepatic in location. The combination of a markedly elevated serum transaminase activity (SGO-T or SGP-T) and a positive EEB-U

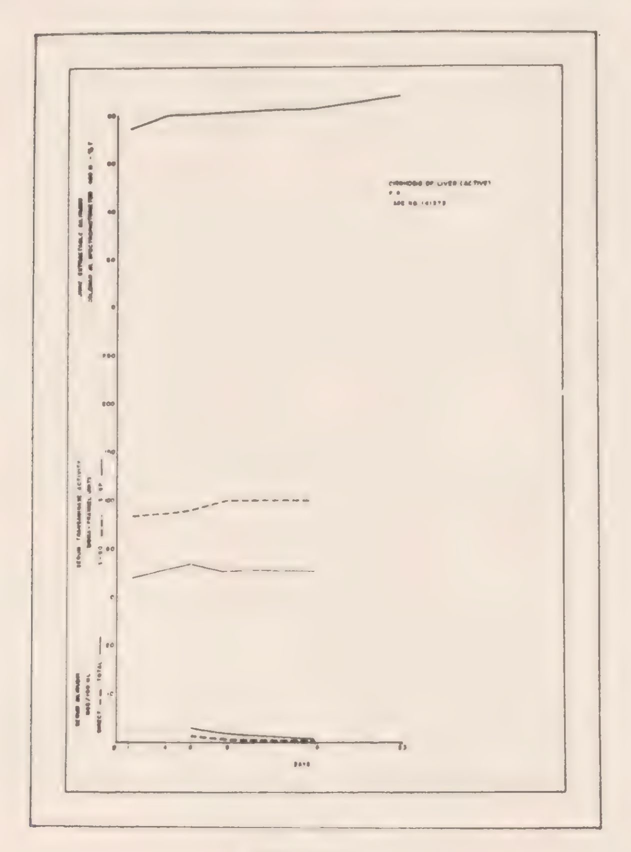


FIG. XII

test has been found in cases of acute hepatitis with an obstructive phase almost exclusively. A rapid change (within one week after the onset of jaundice) from a positive to a negative EEB-U was observed in these cases. Recovery took place in all the cases and the serial determinations of these tests revealed a progressive drop in the serum bilirubin. Acute hepatitis with jaundice in the absence of an obstructive component presented a negative EEB-U test and a markedly elevated serum transaminase activity.

Cases of extrahepatic biliary obstructive jaundice secondary to neoplasms are characterized by a positive EEB-U test, a normal to moderately elevated serum transaminase activity and a sustained hyperbilirubinemia. The EEB-U test changed from positive to negative slowly (one week or longer after onset of jaundice).

One case of pericholangitic hepatitis was found in this study and it presented the picture of obstructive jaundice with slight elevation of serum transaminase activity, sustained hyperbilirubinemia and a positive EEB-U test which rapidly changed to nor-

mal levels. The differentiation of this case from those of extrahepatic biliary obstruction is most difficult. Liver biopsy is a possible solution to this problem, as well as the use of adrenal corticoids.

For active cirrhosis of the liver the tendency has been to present a normal or variable elevation in transaminase activity; a gradually subsiding hyperbilirubinemia, and a negative EEB-U test with occasional positive results on serial determinations.

Disease processes as choledocholithiasis, cholangitis, hepatomas, metastatic liver neoplasms, etc., should be studied with these tests for a more complete evaluation of their usefulness and limitations.

The simplicity of the procedures plus the fact that they are not expensive are added factors favoring their adoption, if more data confirms the results presented.

CONCLUSION

The data analyzed is not sufficient to warrant any final conclusions at this point. Nevertheless, the findings presented suggest that this combination of tests, serum transaminase activity and ether-extractable bilirubin in urine plus the serum bilirubin partition, when performed serially are potentially useful as an aid in the differential diagnosis of jaundice if properly used and interpreted.

ACKNOWLEDGEMENT

The authors acknowledge with pleasure the cooperation of Dr. Juan M. Bertrán and Dr. Roque Nido, from the Surgical Department of Bayamón District Hospital, who performed the liver biopsies reported in this study.

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THE DUBIN-JOHNSON SYNDROME OR CHRONIC IDIOPATHIC JAUNDICE

REPORT OF A CASE

*CHARLES LEE, M.D.,** RAMON A. SIFRE, M.D., LORENZO GALINDO, M.D., and JOSE A. RULLÁN, M.D.*

In the study of cases with jaundice, the clinician occasionally encounters instances where the jaundice does not fall into any of the common categories. In dealing with the less common and more obscure cases of jaundice, we will find a group of cases where the elevation in the values of the serum bilirubin does not appear to be associated with either hepato-cellular or hemolytic or obstructive processes. Most physicians are aware of the entity known variously as familial nonhemolytic jaundice, constitutional hepatic dysfunction or Gilbert's disease. In this condition we find the familial occurrence of hyperbilirubinemia of the indirect type associated with normal liver function tests. In 1954, Dubin and Johnson[1] from the Armed Forces Institute of Pathology and the Veterans Administration described a new condition which they termed "chronic idiopathic jaundice". Since the original presentation Sprinz and Nelson,[2] Stein,[3] and others[4-10] have reported on additional cases. It is our purpose to report another instance of this syndrome in order to call further attention to its occurrence. It is the first such case reported in Puerto Rico.

CASE REPORT

The subject of our report, C.R.C. (S.J.C.H. No. 56-16571) is a 28 year old white male who was admitted to the Medical Department of the San Juan City Hospital on October 12, 1956 with complaints of occasional fatty food intolerance and occasional episodes of right upper quadrant pain of slight to moderate intensity of about six months duration. The patient also gave history of having been told he had yellowish sclerae by several physicians, but had never noticed this himself. He was first told about his icteric sclerae in 1950 by a physician in New Jersey when the patient developed an eye infection. In January 1951, he was admitted to a New Jersey Hospital for his eye trouble which was diagnosed as chorioretinitis. During this admission he was also

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found to have icteric sclerae and an icterus index of 26.2. FBS, NPN, CBC, ESR, serology, urinalysis were all within normal limits and Widal agglutinations were also negative. The patient was finally discharged with the diagnosis of chorioretinitis and infectious hepatitis.

The patient did well until six months prior to his hospitalization at the S.J.C.H., at which time he started developing occasional moderate right upper quadrant pain especially after fatty meals, without any other accompanying symptom. He was seen by a local M.D. who prescribed a low fat diet and Hepa-Desichol and the patient improved although he still felt the pain occasionally. Laboratory tests performed at this time revealed: BSP 8.3% after 45 minutes; Hanger's test of 2 plus and 4 plus; TSP 6.1 Gm.% with 4.14 GM.% Albumin and 1.96 Gm.% Globulin; Bilirubin 1.4 mgm. % direct and 1.0 mgm. % indirect. Sedimentation rate and hematocrit were normal.

On October 1956 he was seen at the San Juan City Hospital and admitted when he was noticed to have moderately icteric sclerae. The physical examination was essentially negative except for the slight icterus and moderate RUQ tenderness on palpation. No hepato or splenomegaly were noted. The past history was negative except for the chorioretinitis and the chronic latent jaundice. There was no family history of jaundice.

Laboratory studies showed: CBC, prothrombin time, bleeding and clotting time, platelet count, Coomb's test, reticulocyte, count, red cell fragility test, alkaline phosphatase, serum phosphorus, Hanger's, thymol turbidity, $ZnSO_4$ turbidity, Vitamin A absorption, serum iron and iron tolerance studies were all within normal limits. Total serum protein was 8.4 Gm.% with 4.4 Gm.% Albumin and 4.0 Gm.% Globulin. The serum protein electrophoretic pattern was normal. BSP retention after 45 minutes was only 6%. The serum bilirubin on admission was 0.84 mgm.% direct and 0.86 mgm % indirect; before discharge the values were 1.2 mgm % direct and 1.2 mgm % indirect. Examination of feces was negative for occult blood and for bile, and positive for urobilinogen. Urinalysis was normal and the urine was found to be positive for bile and traces of urobilinogen were found at 1 10 dilution. A cholecystogram was reported as showing "considerably less than usual concentrating ability in the gall bladder after doubly intensified examination". By duodenal drainage only a light yellow bile was obtained even after stimulation with $MgSO_4$; no crystals or pigment were found in the microscopic examination of the bile. A liver biopsy was done and the results will be described below.

Our patient was again hospitalized seven months later due to infectious parotitis and secondary orchitis. Several studies were

repeated with the following results: CBC, urinalysis and serology were negative, Thymol turbidity 2.6 units, Hanger's test plus-minus and one plus, bilirubin 1.3 mgm % direct and 0.6 mgm % indirect. A serum amylase study done on admission was 328.8 mgm %. As can be seen by these results, the liver condition is stable and did not appear to be jeopardized by his infectious disease.

Pathologic Report:

Pathologic findings on biopsy of liver (Figure 1)—Microscopically, there was no parenchymal alteration in the liver tissue. Slight lymphoid cell infiltration of the portal fields was noted. The most prominent histological change was the presence of a coarsely granular brown pigment in the cytoplasm of the liver cells. The pigment deposits were more dense and prominent in the center of the hepatic lobules. The Kupfer cells were essentially normal.

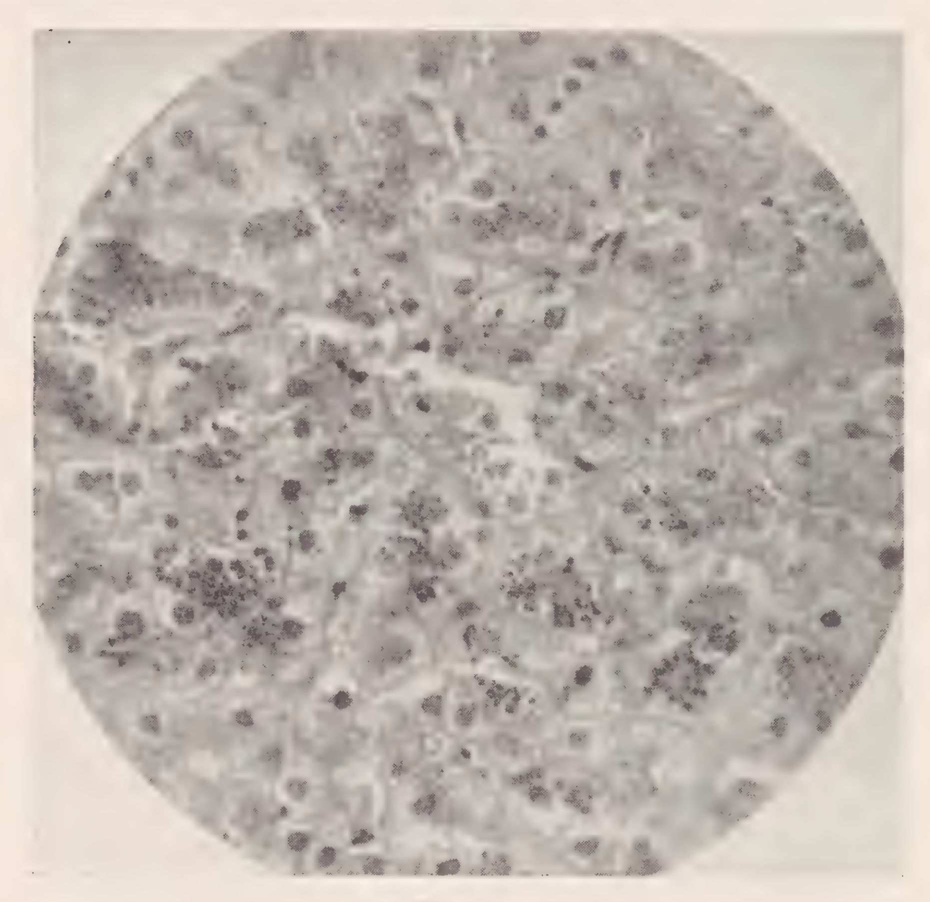


FIG. I

Histochemical Studies: All studies were carried out on paraffin tissue sections.

The following table presents the main findings in comparison with other published cases:

TABLE I: DUBIN-JOHNSON SYNDROME
COMPARATIVE PROPERTIES OF PIGMENTS

TEST OR PROPERTY	THIS CASE (S.56-2893)	PUBLISHED CASES
Color	Light Brown	Yellowish Brown
Basophilia	Positive	Positive
Under Polarized Light	Isotropic	Isotropic
Action of Solvents (alcohol, ether)	Resistant	Resistant
Stain for Bilirubin	Negative	Negative
Stain for Iron	Negative	Negative
Acid Fast	Slight Positive	Positive or Negative
Reduction Ferric Ferricyanide	Negative	Positive
Performic Acid - Schiff (PFAS)	Negative	Usually Positive
Sudan Black B	Not Performed	Usually Positive

Dubin and Johnson[1] considered this pigment as a lipochrome-like substance. Brown and Shnitka[8] believed that this pigment should be classed as a lipofuscin substance. Acording to Pearse, lipofuscins are oxidation products of lipids, and he expresses the natural history of these substances with the following graphic:

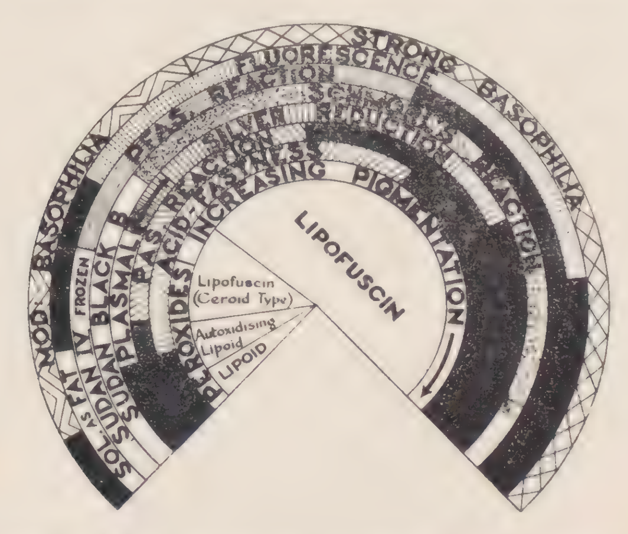


Fig. 2. Reproduced thru the courtesy of Pearse, Histochemistry, p. 262, J and A. Churchill Ltd., Publishers, London, England.

As oxidation of the lipid precursors increases, their chemical and physical properties change and pigmentation usually becomes stronger with this process. Since the degree of oxidation is variable, it is easily explained why the staining properties of lipofuscins show fluctuation between individual cases and even differences in staining of pigment granules in the same histological slide. The case on hand was subjected to only a few staining procedures due to the scarcity of the material. The underlying cause of this disease remains unknown up to the present time.

DISCUSSION

In our patient jaundice became manifest at age 22, 6 years prior to admission to S.J.C.H. In the previously reported cases the onset of jaundice has varied from birth to late old age, although the highest number of cases were in their twenties. It is impossible to determine the exact age of onset of our patient's jaundice since he may have had it for a long time prior to the time when it was called to his attention.

Previously reported cases give no familial history of icterus. This was also true in our case. On the other hand, in constitutional hepatic dysfunction a familial occurrence is almost the rule.

Our patient complained of frequent episodes of right upper quadrant abdominal pain of dull, aching character and of short duration. The presence of pain is only occasionally observed in constitutional hepatic dysfunction, being however frequently present in the Dubin-Johnson group. In the 12 patients of their original series, pain was noted in 11 out of 12 cases. Weakness, fatigability, anorexia, nausea and/or vomiting were not present in our case.

No evidence of hemolysis has been uncovered by other authors. Our patient also gave negative results to the usual tests for hemolysis with lack of anemia, spherocytosis, reticulocytosis, increased osmotic fragility, or positivity of Coomb's test. The urine showed normal amounts of bile and urobilinogen. Fecal urobilinogen determinations were not performed, neither were red cell survival studies.

The serum bilirubin levels were fluctuating with 0.84 mgm %, 1 minute direct reacting bilirubin and 0.86 mgm % indirect on one occasion and 1.2 mgm % direct and 1.2 mgm % indirect the next time. Seven months later, in the second S.J.C.H. admission of our patient, the serum bilirubin values were 1.3 mgm %, 1 minute direct reacting bilirubin and 0.6 mgm % indirect. The very definite elevation of the direct bilirubin is in contrast with the cases of Gilbert's disease where the elevation takes place in the indirect fraction.

The bromsulphalein determination, using the 5 mgm. kilo dose, with a specimen obtained after 45 minutes gave a slight elevation (8.3%) on one occasion and was within normal limits (6%) later on. Abnormal BSP detention has been frequently though not universally found in the other cases.

The cephalin flocculation test was definitely abnormal at one time (2 plus, 4 plus) and normal later on (plus-minus, one plus).

The thymol turbidity was consistently abnormal (6.3, 6.6) but the zinc sulfate turbidity gave a normal value of 8.8.

The alkaline phosphatase gave normal values as well as the prothrombin determinations. The serum proteins fell within normal limits.

The total serum cholesterol was elevated to 311 mgm % with a low ester percentage of 52%.

The serum oxaloacetic and pyruvic transaminase determinations were both normal.

An oral cholecystography with a double dosage gave only faint visualization of the gall bladder shadow.

Failure of visualization of the gall bladder has been shown to occur by previous authors.

At the time of liver biopsy we were very surprised by the gross appearance of the specimen obtained in that it was of a dark green-black color. Other authors have commented on this finding.

Thus we see that our case conforms with the previously reported cases.

The establishment of a diagnosis of the Dubin-Johnson Syndrome would appear to be of more than just academic interest. In cases of chronic continuous or intermittent jaundice the attending physician may be tempted to offer a poor prognosis because of an erroneous impression of chronic hepatitis. However, if liver biopsy is performed and the presence of the typical pigmentation confirmed, then the prognosis can be said to be excellent since it appears from all reports that there is no interference with the normal activities or lifespan of individuals affected with this condition. Again the presence of jaundice over a prolonged period of time together with the finding of non-visualization or poor visualization of the gall bladder at the time of cholecystography could make one suspect the possibility of extrahepatic obstructive jaundice. Recognition of this new syndrome may prevent unnecessary laparotomy in such cases.

SUMMARY

1—A case of the Dubin-Johnson syndrome is reported. This is the first case reported in Puerto Rico, although other authors

have reported two additional instances of this condition in Puerto-rican soldiers.

2—The case fulfilled the postulates of increased direct-reacting bilirubin, abnormal liver flocculation tests, abnormal BSP retention, poor visualization of the gall bladder after double intensified oral dose, and the gross and microscopic presence of the characteristic idiopathic pigmentation within the liver cells.

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ARTERIAL EMBOLIZATION: MODERN CONCEPTS OF MANAGEMENT

(CASE REPORT OF SUCCESSIVE AORTIC "SADDLE" AND FEMORAL EMBOLECTOMIES IN AN ADVANCED CARDIAC PATIENT)

*JOSE S. LICHA, M.D. and JOSE M. TORRES-GÓMEZ, M.D.**

Embolization into the aorta and peripheral arteries represents a dramatic event which has been associated with a high morbidity and mortality.[10,16] Modern concepts of surgical therapy which include rapid intervention, have definitely saved limbs and lives.[3,4,5,16,28] Yet, according to Pool et al,[15] up to 1952, only thirty three (33) successful aortic embolectomies had been performed.

We believe that the presentation of this case of successive multiple aortic and peripheral arterial embolizations is justified not only to add another successful one to a rather limited registered number,[7,8,16,18,19,28] but also to use it as a means to stress modern concepts of management and to announce it as the first successful case of aortic embolectomy performed in Puerto Rico.

CASE REPORT

A 68 year old white widow was admitted to Doctors' Hospital on March 29, 1957 with gross cardiac decompensation, manifested



Figure I

* From Doctor's Hospital, Santurce, P. R.

by orthopnea, rales, tender hepatomegaly, ankle edema, tachycardia, and cardiomegaly. (Fig. 1) A known case of hypertensive and arteriosclerotic heart disease under treatment with digitalis and diuretics for the past 2½ years, her blood pressure had ranged from 164/120 to 140/100 and her pulse had been regular. A Grade I-II systolic murmur, heard at the apex, had been interpreted to reflect the presence of a mild degree of mitral insufficiency secondary to left ventricular enlargement. Difficulty in keeping her adequately compensated had already been evidenced in a previous hospital admission in January 1957 and in office visits in February and March 1957, when restrictions in her diet and activity, and increases in digitalis and/or diuretic dosages were required.

Her state of decompensation was improving gradually under the usual methods of therapy (digitalis, diuretics, bed rest, low salt diet, etc.) when on the fourth hospital day (4/2/57) the patient complained of acute pain over the right thigh and numbness over the entire extremity. Shortly after, the pain shifted to her low mid-abdomen and radiated towards the right hip and knee. Though, at first, only the right leg seemed to be affected, by this time both lower extremities were cold and showed a marked diminution in sensation to pain and touch. No pulsations were felt in either femoral artery or anywhere else in either leg. As an acute aortic occlusion was suspected, a surgical consultation was requested. The consultant's impression was that of a saddle embolus at the aortic bifurcation. Though complete cardiac compensation had not yet been achieved, it was decided to operate in view of the grave complications that severe ischemia could provoke in her lower extremities.

Under endotracheal cyclopropane anesthesia, a saddle embolus was evacuated from the aorta 9 hours after the onset of symptoms, and regular peripheral pulses were adequately felt right after the conclusion of the operation.

On the following day, 4/3/57, all pulses were felt with the exception of those over the right posterior tibial and dorsalis pedis arteries. The right foot felt cool in relation to the left one but normal tactile and pain sensation had returned to both legs. Her convalescence was uncomplicated until the fifth post-operative day when an irregularity was noted in her pulse and an electrocardiogram revealed auricular fibrillation. (Fig. 2) Anticoagulant therapy was started at this time with the hope of converting the fibrillation to regular sinus rhythm afterwards.

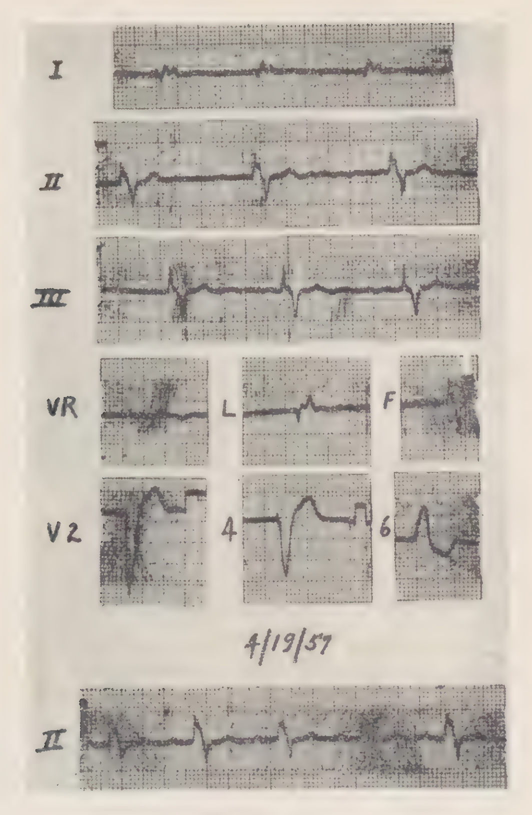


Figure II

Three days later (4 10 57), the patient developed unexplained cold sweats and her right radial pulse became barely palpable. Three hours later the left popliteal pulse could not be felt. The patient had complained of pain over the left thigh and the leg was already turning cold and cyanotic when the surgeon was consulted again. Three and a half hours after the onset of symptoms, a left femoral artery embolectomy and a stripping periarterial sympathectomy were performed. All pulses in the left lower extremity were regained immediately following the operation. Though the patient had been on dicumarol, Vitamin K_1Oxide was not used because her prothrombin time had not yet reached significantly elevated values. The dicumarol was then increased and two days after surgical intervention the patient's prothrombin time was 30 seconds while the control was 13 seconds. There were no bleeding complications post-operatively.

No surgical therapy was required for the embolus to the right arm because of the evident adequacy of its collateral circulation. On 4 11 57, the first day following the left femoral embolectomy, quinidine was prescribed, and the auricular fibrillation converted

to regular sinus rhythm within 24 hours. (Fig. 2) Associated medications received all along included digitalis orally and or intramuscularly, Neohydrin orally, different forms of low salt diets, papaverine parenterally, small doses of sedatives, and antibiotics. No further embolic complications occurred after the arrhythmia was corrected. The patient was maintained on 0.2 grams of quinidine and 100 mgms. of dicumarol daily. The prothrombin time varied between 32-40 seconds with a control of 13. By 4/13/57, three days after surgery had been performed on the left femoral artery, she was ambulating and finally discharged on 4/20/57, 3 weeks after admission.

While at home, the patient remained active doing all types of domestic work. She complained for a short time of aches on and off related to her legs and toes but repeated examinations on follow-up office visits did not reveal any ischemic signs. The toenails remained intact. There was no loss of tissue. Gangrenous changes were never evident. The pulse remained regular, always felt at both femoral and radial arteries, but intermittently felt at the popliteal and ankle levels.

By June 1, 1957 she developed hematuria. Intravenous pyelography showed normal renal structure and function. Dicumarol was discontinued but as hematuria persisted. 50 mgms. of Vitamin K_1 Oxide were given intravenously. On the following day the urine was normal. One month later dicumarol was resumed but as she developed hematuria again, the drug was discontinued permanently. By August, the patient began to show signs of decompensation such as gallop rhythm, dyspnea on exertion, rales, and hepatomegaly. The dose of digitalis was increased but no relief was obtained.

It was then discovered that she had discontinued quinidine and had gone beyond the limits of activity that her heart disease permitted. Once more hospitalization was required on August 20, 1957. An electrocardiogram confirmed the clinical impression that fibrillation was present again. Changes in the configuration of the QRS complex strongly suggested the occurrence of a myocardial infarction. On August 23, 1957, 3 days after admission, while the patient was gradually regaining cardiac compensation, she suddenly developed difficulty in breathing, cyanosis, and loss of blood pressure, dying a few minutes after the onset of this episode. (Fig. 3) Though permission for autopsy was not granted, it is our impression that this patient died of another attack of coronary thrombosis, 4½ months after the performance of successful saddle embolectomy.

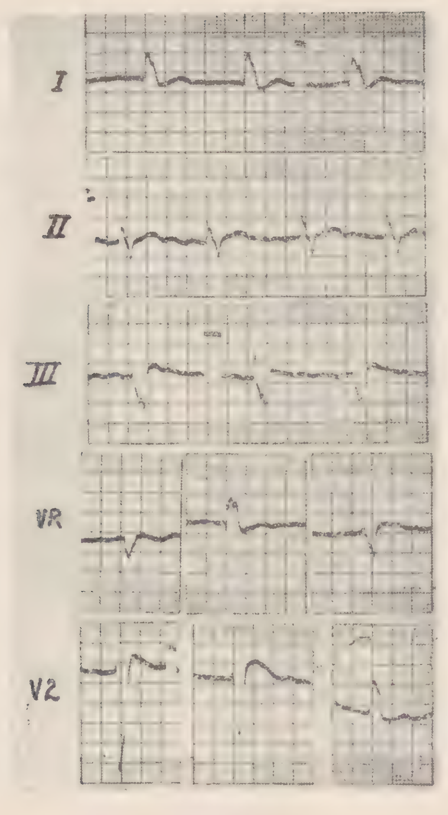


Figure III

A. Incidence

Aortic embolizations comprise 4-8% of all peripheral arterial emboli. Up to 1950 only 193 saddle embolectomies had been reported with 26 successful cases.[10] All but five of these 26 cases had been operated upon within 6 hrs. In 1952 Pool reported a total of 33 successful cases gathered from the literature.[15] Around 75-85%[10,16] of remaining emboli find their way into the main arterial channels of the lower extremities, 55% lodging in the femoral arteries where the superficial location of these vessels makes surgical access an easy matter.

B. Cause

About 90-95% of arterial emboli have their origin in the heart as clots in the auricle in the presence of auricular fibrillation, or as mural thrombi in myocardial infarction.[7,10,16] In our case we believe the cause to be myocardial infarction with mural thrombus detachment and lodgement of embolus at the aortic bifurcation in the first incident. Subsequently, the initiation of auricular fibri-

llation, contributed with other embolizations to the left femoral and right brachial arteries.

C. Recurrence

The tendency for recurrent embolization is common[6] and was found to occur in 39.5% of 200 patients reported by Warren et al,[16] 53.7% of cases occurring in rheumatic fibrillators and 2.5% requiring more than one embolectomy.

D. Time

Prompt diagnosis of a case of embolization with rapid intervention, preferably within a limit of 6 hrs., is the best guarantee of success. Procrastination only spells disaster in the great majority of cases.

95% survival was reported in 19 extremities treated within 6 hrs. by Shumacker et al[1] while 50% survival was reported in 8 extremities treated within 6-13 hrs. Other workers[6,12,21,22] attest to the importance of early detection and treatment.

E. Pathogenesis

An understanding of the pathogenesis of the sudden occlusion of a main artery by an embolus is mandatory if prompt diagnosis and proper management are to follow. Sudden occlusion leads to anoxia of tissues in the distal portion with vasospasm in the remaining collateral vessels. Vasospasm is dependent upon the sympathetic nervous system as the efferent arm of a reflex are having its focus of irritation at the occluded portion of artery. If vasospasm persists, thrombosis propagating proximally and distally from the embolus, eventually affects the collaterals in the process,[25] thus, jeopardizing the chances of limb survival. Yet, it is amazing how far the thrombotic process may progress in a main arterial channel without involving the patency of collaterals. This fact is advanced as an important reason for aggressive surgical intervention in "late-cases" with good hopes of therapeutic success.[3,4,12]

F. Diagnosis

Early therapy can only follow early diagnosis. In this case, pain, pallor, pulselessness and lowered skin temperature of the affected extremities spelled obstruction of main arterial channels—aorta in the first instance and left femoral and right brachial artery in the second episode. This complex of symptoms and signs in a known arteriosclerotic cardiac case with coronary throm-

bosis with known previously patent arteries served to cement the diagnosis in the mind of the attending cardiologist, requiring immediate surgical consultation.

The oscillometer and other diagnostic measures such as arteriography may be utilized in establishing a diagnosis, but most if not all cases are readily diagnosed by the facts offered by a careful history and physical examination. In this regard Albright and co-workers[10] stress the importance of the 5Ps: pain, pallor, paralysis, pulselessness and paresthesia.

G. Treatment

The issue of conservative versus surgical treatment should have no place in any discussion on this subject. It is only through the proper evaluation of every case that a decision should be made, as true in all problems in medicine. In our case both conservative and surgical procedures were utilized. Conservative treatment for the right brachial embolization was prescribed because on examination of the right upper extremity adequate collateral circulation was found as evidenced by practically no symptoms or signs of acute arterial occlusion outside of a greatly diminished right radial pulsations. Veal and co-workers[17] contend that embolism of the brachial artery can be treated with success under a conservative regimen, rarely requiring embolectomy.

The use of sympathetic blocks, anticoagulants, alternating venous occlusion, sympathetic ganglionectomies, peri-arterial sympathectomies (utilized by us as an adjunct to the femoral embolectomy) are conservative measures which have their place as adjuncts in the modern treatment of arterial embolizations. They should be weighed in the light of a careful analysis of the case at hand so that the golden opportunity of saving a limb is not disregarded thru the acceptance of apparently safer but not adequate medical or surgical measures. The only yardstick of success in the treatment of arterial embolization, is a usable, asymptomatic limb and not merely one that has "survived".

Good flow of blood in a limb is considered of far greater importance in preventing post operative thrombosis than is anticoagulant therapy although there is no reason why these substances should not be used judiciously if deemed necessary. Warren and co-workers[16] report in a series of 20 patients treated with long term anticoagulants 4 bleeding complications and 2 emboli during adequately induced hypoprothrombinemia. Yet they still considered it reasonable to use anticoagulants where the risk of further embolism is great. Two of their cases had embolism two weeks following cessation of therapy.

Aortic saddle embolization, on the other hand, should be treated surgically as soon as a diagnosis is made preferably within 6 hours unless gangrene has supervened in the affected extremities or the patient is meribund. Delay can only spell disaster.[11] Of 10 cases of aortic saddle embolus treated conservatively by De W[illegible][21] 7 died and 3 developed gangrene of 1 limb. Intervention, 9 hours after the occlusion in our case, led to complete success as evidenced by an immediate restoration of peripheral pulsations following left common iliac arteriotomy and removal of the aortic embolus by "milking". (Fig. 4)

EMBOLECTOMY (AORTIC AND FEMORAL)

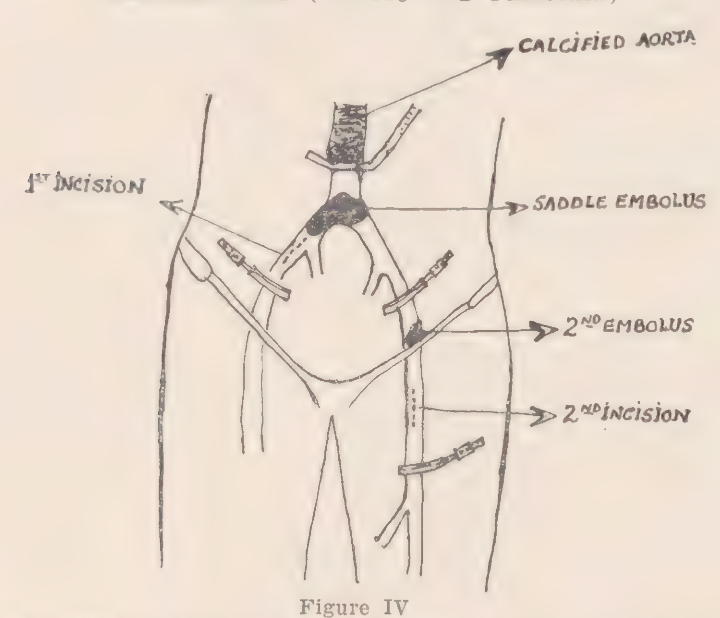


Figure IV

It would have been technically difficult and unduly hazardous to perform an aortotomy in accomplishing saddle embolectomy because the abdominal aorta had a stony hard consistency down to about 1" from the bifurcation. Right iliac arteriotomy with milking of the embolus thru the incision proved to be an easy surgical manoeuver accomplishing its purpose satisfactorily. The embolus was removed in its entirety as proved by the presence of two smooth ends and the free flow of blood from proximal and distal portions of the artery on the release of the occluding clamps. This technique was utilized by Linton in 1915[26] and more recently recommended by Gordon[9] who believes it preferable to an aortotomy and by Weisman et al[28] who employed it with success in two cases.

In accomplishing the left common femoral arteriotomy for removal of an embolus lodged in the vessel at its upper end free flow of blood was also immediately obtained from the proximal and distal ends of the vessel confirming arterial patency above

and below the previous site of obstruction. This could be accomplished because no propagation of thrombus had occurred due to early intervention. When a distal thrombus is present, there are various surgical manoeuvers that should and can be carried out either singly or in combination as follows:

(1) Suction applied to urethral or other type of catheter introduced thru an arteriotomy proximally and distally with or without the introduction of heparinzed normal saline solution for lavage. This procedure has been reported as used by Lehrman in 1930,[2] by Harkins in 1932,[27] and by others more recently.[11]

(2) "Milking" of the distal thrombus towards the arteriotomy incision by means of an elastic bandage which is applied distally and wrapped toward the arteriotomy site. This method was utilized successfully by Keeley et al[3] in 1951 and since then its use has been reported by other workers.[12]

(3) Retrograde "Flush Technic". This is the procedure of flushing the affected artery at a site distal to the obstruction with the intent of pushing the propagated distal thrombus retrogradely thru another arteriotomy site at or near the point of obstruction. Lerman in 1930[2] and later on Olwin in 1953,[3, 11] Shaw in 1956[1] and more recently in 1957 De Bakey[12] have utilized enthusiastically this technic and all recommend it as the one most uniformly successful in their hands. No doubt this is the method most widely accepted in the present day surgical management of the complication of distal thrombus propagation in embolization to arteries. (Fig. No. 5)

RETROGRADE FLUSH TECHNIC



Figure V

It is fortunate that the smaller branches of thrombosed arteries are not invaded by clots in many instances as shown by immediate back bleeding from small branches following evacuations of clots during the successful acomplishment of the "flush" manoeuver. It is thus possible to salvage limbs in many a victim of a "fatal case" of embolization as proven by the proponents of the method. The only two requirements for success according to Dr. De Bakey[12] are:

(1) Patent artery prior to embolization.

(2) A viable part in the region supplied by the artery rather than opportunity expressed in terms of time.

Successful cases by the "flush technique" are cited from 5 days[12] to as long as 13 days[16] post embolization. Yet, best results are still obtained in earlier cases where the necessity for the "flush" or other manoeuvers is obviated.

In a review of present day opinion on the management of arterial embolism whether located in the aorta[9,10,15,17,26,27,28] or other accessible peripheral arteries (iliac,[27] femoral,[8,27] popliteal,[11,20] brachial and superior mesenteric[23]) the general tendency is towards embolectomy as the treatment of choice—whether the case is early or late—as long as there is possibility of reversing the nutritional status of the involved limb by the utilization of the various technical surgical measures already mentioned particularly "retrograde flushing" of the distally propagated thrombus.

A 95% limb survival with aggressive surgical embolectomy performed within the golden time limit of 6 hrs. is reported by Schumacker.[6] De Bakey[12] in turn has complete salvage in 10 out of 12 limbs (one 5 days after the accident) by the utilization of embolectomy with the "retrograde flush" technic. Warren and associates[16] report an 83.3% overall limb survival in embolectomized cases.

Conservative treatment is advocated only in those cases where obviously the involved limb shows adequate circulation with or without the benefit of sympathetic block, and by some authors in the management of embolisms of the popliteal[16] and brachial artery.[17]

SUMMARY

(1) A case is presented of an advanced cardiac with multiple successive arterial embolizations (aortic, femoral and brachial) managed successfully according to modern precepts of treatment by prompt and coordinated action on the part of internist and surgeon.

(2) Brief discussion of incidence, cause, pathophysiology, and diagnosis of arterial embolization is included.

(3) Conservative and adjunct methods of treatment are enumerated and present-day opinion on their proper utilization briefly outlined.

(4) A technic for aortic embolectomy is described utilizing a common iliac arteriotomy as a substitute for aortotomy in the case where technical difficulty is anticipated in performing the latter

(5) A brief description is given of the different surgical manoeuvers utilized in dealing successfully with late cases of arterial embolization with propagated thrombus. Special emphasis was given to the "retrograde flush" technic as the most efficient to date in the modern management of this dreaded complication of arterial embolism.

(6) Definite stress is given to the fact that embolectomy is the treatment of choice for most arterial embolizations early or late as evidenced by a consensus of present day opinion.

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NEWER CONCEPTS IN THE TREATMENT OF METASTATIC OCULAR CARCINOMA

*ROBERTO BUXEDA, M.D.**

Although carcinoma is the most common secondary tumor affecting the inner eye, nevertheless its occurrence is rare. This is probably due to the fact that the metastases are blood-born and since the ophthalmic artery leaves the internal carotid at right angles, it is not readily entered; it is easier for malignant emboli coming by this route to travel straight on and lodge themselves in the minute circulation of the brain and meninges. It is very likely, however, that such metastases are considerably more frequent that the literature would indicate, for in most of the cases the patient is already gravely ill and the clinical picture is dominated by his general condition while he himself does not notice what may be a minor defect in one eye.

The posterior region of the choroid is the most frequently involved portion of the eye, especially its temporal side near the macula where the short ciliary arteries are most numerous and largest while involvement of the ciliary body and iris is rare. The reason for this is that it is easier for the vast majority of emboli to travel up the 20 odd short posterior ciliary arteries rather than the 2 long posterior or the five anterior arteries. Since such emboli are usually liberated in numbers, multiple tumors are not uncommon and in 20.8% of the cases both eyes are affected, usually not simultaneously, but rather one following the other. The left eye is more commonly affected than the right, this incidence being comparable to the great frequency of left-sided cerebral and ocular embolism which is doubtless due to the more direct pathway by the left carotid artery.

Metastatic ocular carcinoma usually occurs from primary carcinoma in the female breast. A unique case secondary to carcinoma of the male breast was reported by Giri.[1] The second most common site of the primary growth is the lungs, then the alimentary tract and thereafter the thyroid and the liver. Rare sites are the prostate, the ovary and the parotid gland. It is somewhat difficult to account for the preponderance of breast tumors in the etiology, since, while they are responsible for over 50% of uveal metastases, their general incidence is only 13.5%, while carcinoma of the stomach with a relative frequency of 36.5% gives rise only to 3.3% of uveal metastases. Ask[2] suggested that this was pro-

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* From the Department of Ophthalmology School of Medicine, University of P. R., San Juan, P. R. Read at the Annual Meeting, P. R. Chapter, American College of Surgeons, March 1, 1958, San Juan, P. R.

bably due to a varying predilection for hematogenous metastases in the primary growth, a factor presumed to be high in breast tumors; but it is probable also that the uvea provides more suitable nutritional conditions for some tumor cells than for others.

Due to the preponderance of breast cancer as the causal lesion, metastatic ocular tumors are commoner in females than in males for obvious reasons. Most cases occur from 40-50 years of age, and below 30 and above 70 they are rare.

While intraocular metastases usually occur as part of a general dissemination of the disease, they not uncommonly occur quite early in its evolution. Many cases have been reported in the recent ophthalmological literature in which the visual disturbance was the first symptom noticed by the patient and has led to a systemic investigation in search of the primary growth. Recently in the May issue of the American Journal of Ophthalmology Van Wien and Schoch[3] reported the case of a 71 year old white woman with bilateral choroidal metastatic carcinoma with involvement of the optic nerves in which ocular symptoms occurred eight months before the primary breast tumor could be discovered. Because of extensive retinal detachment no ophthalmoscopic diagnosis could be made. In spite of repeated and thorough physical examinations, the primary carcinoma of the breast was discovered only several months after appearance of the initial eye symptoms. The importance of diagnosing these ocular tumors is therefore obvious. The most common sites in which a primary growth may be latent in this fashion are the gastrointestinal canal, the lungs and the thyroid.

The typical ophthalmoscopic appearance of a metastatic carcinoma of the choroid is somewhat distinctive. The tumor is frequently diagnosable from its appearance alone. Unlike the usual form of a malignant melanoma, it is rarely circumscribed, but rather appears as a flat thickening of the choroid, usually thickest at the posterior pole and thinning off anteriorly, over which there is a shallow retinal detachment. The swelling is of a pale grey color, the surface usually displays a grey mottling, and the edges are not sharply defined. Small hemorrhages are not uncommon on its surface, or vessels of new formation, but the vitreous is rarely disturbed. Increase of growth occurs rapidly, more rapidly than in a flat melanoma, glaucoma is late in appearing, but pain is earlier and more pronounced than in primary tumors and frequently involves the necessity of enucleation. At any time, of course, a large retinal detachment may occur obscuring the details of the clinical picture, and its occurrence, particularly its bilateral occurrence in a carcinomatous patient is always suggestive.

The prognosis in these cases up to the immediate past was invariably considered to be bad; they would always end fatally shortly after discovery of the lesion owing to metastases. Usher,[1] in his review of 110 cases, found that the average duration of life after the ocular lesion has been noted was 8 months, the longest interval being 2 years and the shortest 4 weeks. The great majority of patients, indeed, are already cachectic, and multiple metastases co-exist in many organs of the body.

The treatment used to be therefore, palliative. The attitude of the ophthalmologist in the past when confronted with such cases was one of hopelessness. He therefore, either would do nothing or would occasionally treat the involved eye by irradiation chiefly for moral purposes.

Dunphy[5] has brought up to date recently what the ophthalmologist, the gynecologist, the general surgeon and the neurosurgeon working together are doing to help give hope to these hitherto hopeless cases.

Recently the recognition that certain tumors (especially those of the breast and prostate) can be influenced favorably by the action of hormones has given rise to hopes that many unfortunate individuals may have their lives prolonged and eyesight improved at least temporarily. We have on record several cases of patients with matastatic disease of the eye difinitely benefited by this form of therapy so that it behooves ophthalmologists in general to be familiar with the medical and surgical principles involved.

It has been common knowledge for some time among general surgeons and urologists that androgens or sterilization may temporarily control carcinoma of the breast in many cases, whereas estrogens or castration are definitely beneficial in the therapy of carcinoma of the prostate. However, some curious contradictions began to appear. For example, Stilbestrol, while helping most cases of prostatic carcinoma, occasionally helps cases of carcinoma of the breast also. This is particularly true in patients in the post-menopausal stage of life, whereas in patients in the premenopausal stage of life it may actually make the disease worse.

The reports on this subject in the literature are scant. In 1952 Ellis & Scheie[6] reported marked regression in bilateral choroidal metastases in a case of a woman with cancer of the breast following sterilization by X-ray radiation.

In 1954 Cogan and Kuwabara[7] reported a case of breast cancer with choroidal metastases to one eye, which increased despite testosterone, but regressed markedly with Stilbestrol for a two year period.

This state of confusion has been cleared up to some extent by the work of Huggins[8,9] and his associates. They have shown that after oophorectomy there is a compensating hypertrophy of the adrenals and increased cortical function. Thus, many women continue to excrete extrogenic substances even after surgical castration, but these usually disappear following bilateral adrenalectomy. Similarly, in men castration produces an increase of the 17-ketosteroids in the urine which can be eliminated by bilateral adrenalectomy. This knowledge led to the development of the combination of bilateral oophorectomy and bilateral adrenalectomy with the hope that these operations would eliminate all sources of hormones that might possibly favorably influence the metastatic growth. These patients, of course, require cortisone for the rest of their lives. Of course, not all cases respond favorably because it seems that some tumors are not as hormone dependent as others.

Mr. E. F. King[10] of Moorfields Hospital in London reports the case of a woman, aged 53 years, who, one year after mastectomy for breast cancer, developed a large choroidol metastasis, along with extensive pulmonary involvement. A bilateral oophorectomy and adrenalectomy were performed and there followed a complete disappearance of the choroidal metastasis within three weeks and an almost complete disappearance of the pulmonary involvement within three months. One year later the patient was well. King, also reports the case of another patient with cancer of the breast and choroidal metastasis who has done remarkably well for the past three years, following combined surgery on the ovaries and the adrenals. The choroidal metastasis receded markedly.

Another step in the hormonal control of breast and prostatic carcinoma was taken in 1953 by Luft and Olivecrona,[11] who performed hypophysectomy in an attempt to eradicate all sources of gonadotropic and somatotropic hormones and the removal of different possible growth factors. In this fashion the removal of the pituitary gland might have a greater field of usefulness than adrenalectomy. So far the metastatic breast cancers treated with this type of surgery have responded fairly well and the operation is well tolerated in the hands of capable neurosurgeons. It is indeed probable that metastatic eye lesions will also be controlled by this surgical procedure.

It was hoped that, since the pituitary gland has a melanostimulating hormone, the operation might favorably influence malignant melanomas but this has not proved to be the case in four patients with this tumor occurring in other parts of the body.

The time is coming when we will take a much more hopeful outlook on metastatic carcinoma of the choroid and when we ophthalmologists will get our patients quickly to the general surgeon or the neurosurgeon, who may be able to help them a great deal, at least on a temporary basis.

To conclude, I have presented here some of the newer concepts in the management of metastatic intraocular carcinoma other than by enucleation. This latter procedure is not to be condenmed, but we believe it is wise to remember that we are now in possession of other alternatives, which may be applicable in certain cases. It is the feeling now that with our advancing scientific knowledge, the development of chemotherapy, the use of radioisotopes and the accumulation of knowledge concerning the biological effects of hormones less cancer containing eyes will have to be removed in the future.

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EDITORIAL

EL MEDICO Y LA RELIGION

Frecuentemente se han expresado compañeros nuestros en el sentido de que no hay relación entre la práctica de la medicina y la religión y más aún que deberían de permanecer siempre divorciadas. Nada más lejano de la verdad en nuestro humilde parecer. El cuerpo humano está unido por la mano divina al alma y como médicos no podemos menos que considerar este aspecto de nuestros pacientes si es que queremos practicar el verdadero arte de la medicina.

Todos los sistemas religiosos han considerado la vida terrenal como un paso al más allá; como un tránsito de mucha importancia, con un origen y terminación que dependen absolutamente del Creador.

Corresponde entonces a la Divinidad el dar la vida, el don material más grande que nos otorga nuestro Señor, y el quitarla cuando así lo juzgue conveniente en su sabiduría.

Nosotros los que practicamos la profesión médica tenemos el deber de tratar de conservar esta vida utilizando todos los conocimientos de nuestro arte. Faltaría el médico que no conociera los últimos adelantos en la práctica de su rama específica de la profesión. Faltaría a la moral si no aplicase los procedimientos necesarios para la mejor y más pronta curación del enfermo. Igualmente tiene el deber de prepararse y entrenarse para rendir servicios dentro de su máxima posibilidad sin mediar consideraciones de raza, credo o dinero en sus decisiones médicas.

Por otro lado no cae dentro del marco de acción del médico el determinar quién debe vivir y quién debe morir. Como ya expresado, estas decisiones están fuera de nuestra potestad, pertenecen solo a Dios. Por lo tanto, el aborto a un extremo de la vida y la eutanasia al otro son y deberán ser proscritos por la moral de nuestra profesión.

Si consideramos que la vida es un paso a la existencia infinita en el otro mundo, deber será también nuestro el hacer saber a la familia del enfermo y en algunos casos al enfermo mismo, si fuere necesario, la proximidad de la muerte de manera que pueda prepararse adecuadamente por los métodos prescritos por su religión, cualquiera que ésta fuere, para este paso de vital trascendencia eterna.

Muchos otros son los puntos de convergencia entre nuestra profesión y la religión. Debemos los médicos estar más conscientes de nuestras obligaciones al respecto para así rendir un servicio más completo a nuestros pacientes.

LA LEGISLATURA DE PUERTO RICO RINDE MERECIDO TRIBUTO AL DOCTOR TOM D. SPIES

ESTADO LIBRE ASOCIADO DE PUERTO RICO

Asamblea Legislativa

Nosotros, Julio C. Torres y Néstor Rigual, Secretario del Senado y de la Cámara de Representantes del Estado Libre Asociado de Puerto Rico, respectivamente,

CERTIFICAMOS

Que en sesiones celebradas por la Cámara de Representantes y el Senado del Estado Libre Asociado de Puerto Rico durante la Segunda Sesión Ordinaria de la Tercera Asamblea Legislativa, fué aprobada la resolución concurrente cuyo texto es el siguiente:

RESOLUCION CONCURRENTE

Para expresar el reconocimiento del
Estado Libre Asociado de Puerto Rico al

DR. TOM D. SPIES

por sus descubrimientos médico-científicos que han redundado en beneficio de toda la humanidad, y por la inquietud que ha demostrado en favor de las instituciones hospitalarias puertorriqueñas.

POR CUANTO, el doctor Tom D. Spies, natural del Estado de Tejas, es un especialista en nutrición y con gran desinterés se ha dedicado a buscar soluciones a los problemas nutricionales dentro del campo de la medicina;

POR CUANTO, el doctor Tom D. Spies es profesor de metabolismo y nutrición de la Escuela de Medicina de la Universidad de Northwestern, en Chicago, y jefe de la Clínica de Nutrición del Hospital Hillman, de Birmingham, habiendo recibido, en el año 1939 el premio John Phillip's Memorial, del Colegio Americano de Médicos, y una medalla por servicios distinguidos de la Asociación Médica Americana (1957);

POR CUANTO, en uno de los numerosos viajes que ha realizado a Puerto Rico sufragando los gastos de su propio peculio, para trabajar en la Escuela de Medicina Tropical, el doctor Tom D. Spies trajo a nuestra isla las primeras dosis de ácido fólico y los primeros microgramos de vitamina B_{12} para ser usados como experimentos en los casos de enfermos de esprú, siendo el resultado que esta penosa enfermedad es curable ahora con las referidas drogas.

POR TANTO, resuélvese por la Asamblea Legislativa de Puerto Rico:

PRIMERO: Expresar el reconocimiento del Estado Libre Asociado de Puerto Rico al doctor Tom D. Spies, por sus descubrimientos médicos que han redundado en beneficio de toda la humanidad, y por la inquietud que ha demostrado en favor de las instituciones hospitalarias puertorriqueñas.

SEGUNDO: Que copia certificada de esta resolución sea remitida al eminente galeno,

doctor Tom D. Spies.

Y para remitir al doctor Tom D. Spies, expedimos esta certificación que firmamos en nuestras oficinas del Capitolio, en San Juan, Puerto Rico, a los 3 días del mes de marzo de mil novecientos cincuenta y ocho.

Julio Torres
Secretario,
Senado de Puerto Rico

Néstor Rigual
Secretario,
Cámara de Representantes

Vo. Bo.

Samuel R. Quiñones
Presidente,
Senado de Puerto Rico

Vo. Bo.

E. Ramos Antonini
Presidente,
Cámara de Representantes

LA ASOCIACION MEDICA DEL DISTRITO SUR, EN ASAMBLEA GENERAL CELEBRADA EN PONCE, PUERTO RICO A LOS CATORCE DIAS DEL MES DE DICIEMBRE DE MIL NOVECIENTOS CINCUENTA Y SIETE, ADOPTA POR UNANIMIDAD LA SIGUIENTE

— RESOLUCION —

POR CUANTO: La Asociación Médica de Puerto Rico fué fundada hace medio siglo con los firmes propósitos de promover y mantener los más altos cánones de ética profesional entre sus asociados y colocar y preservar en elevado nivel científico y humanitario la práctica de la medicina en nuestro país, de suerte que pudiera cumplir a cabalidad con la finalidad para la que fué creada: velar por la salud de nuestro pueblo.

POR CUANTO: La posición de Presidente de nuestra institución requiere ciencia, tacto, abnegación y una especial dedicación para cumplir de manera eficaz y provechosa con los postulados que le dieron vida.

POR CUANTO: En toda su vida profesional y particularmente durante su cargo como Presidente de la Asociación, el distinguido compañero Dr. Guillermo Picó, defendió con el más sincero fervor los mejores intereses de la clase médica y luchó por el bienestar del país y de todos los puertorriqueños.

POR CUANTO: El Dr. Guillermo Picó ejerció su cargo con la mayor dedicación y ejemplaridad sin importarle todos los sacrificios personales que le fueron requeridos, haciendo consecuentemente, honor a su magnífica historia profesional y a su arraigado sentido del cumplimiento del deber.

POR TANTO: RESUELVASE como por la presente se resuelve por esta Asociación del Distrito Sur de la Asociación Médica testimoniar su más sincero agradecimiento al Dr. Guillermo Picó por la espléndida labor realizada durante su incumbencia; y el más hondo y sentido reconocimiento por su esfuerzo y dedicación tan ampliamente manifestados durante el año 1957.

RESUELVASE además, que copias de esta resolución sean enviadas a las Secretarías de la Asociación Médica de Puerto Rico, de sus Asociaciones componentes de Distrito y a la Prensa de Puerto Rico.

Y para que así conste firmamos la presente a los 14 días del mes de diciembre de mil novecientos cincuenta y siete.

Carlos F. Jiménez-Torres, M.D.
Presidente
Asociación Médica del Distrito Sur

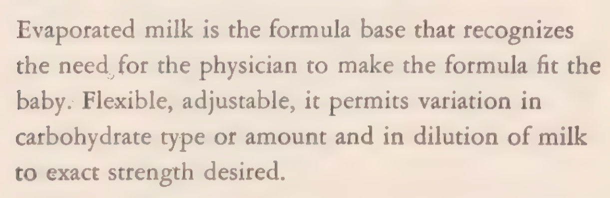
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ASOCIACION MEDICA DE PUERTO RICO

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ASAMBLEA ANUAL

Noviembre 18-22, 1958

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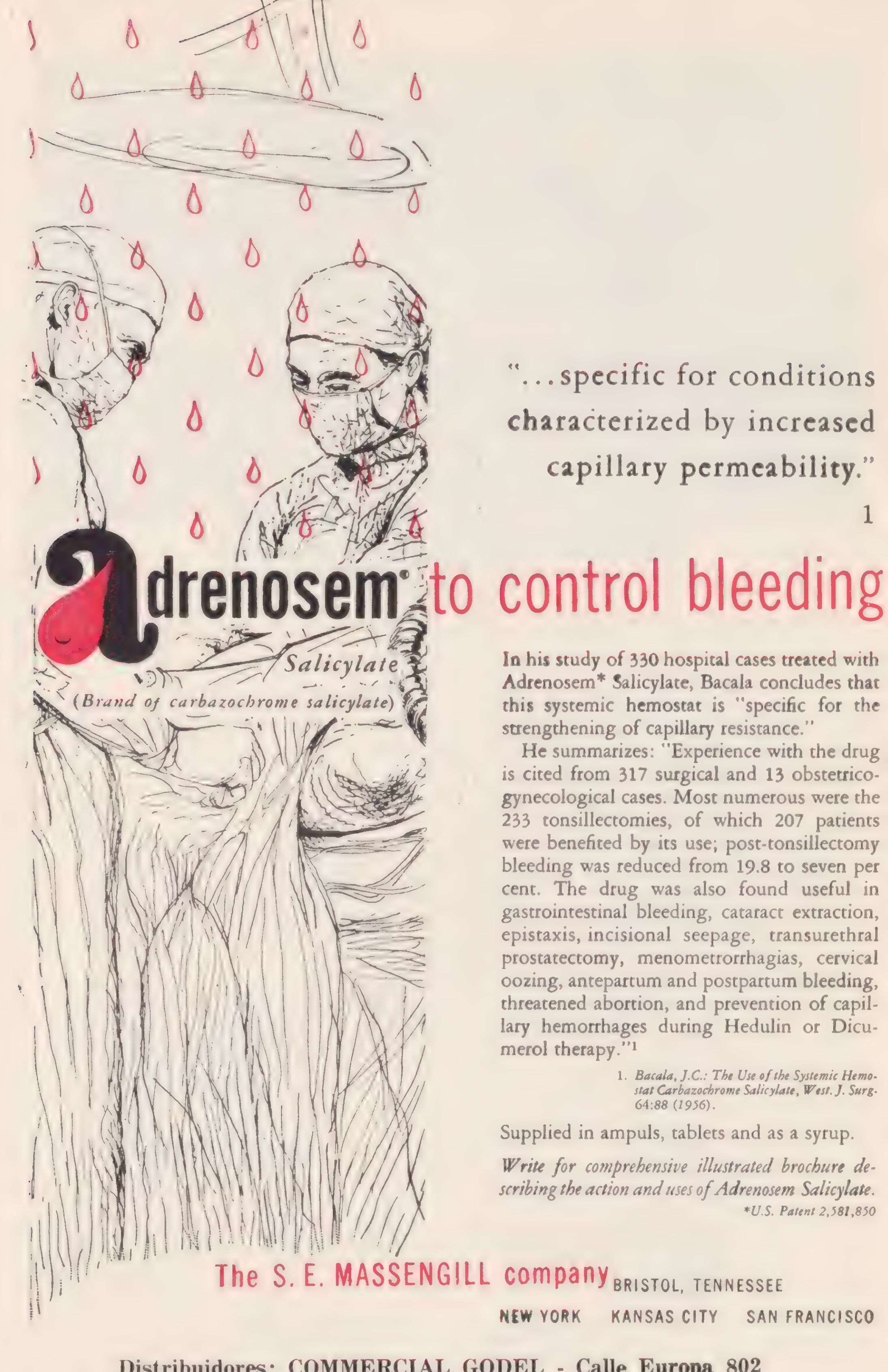
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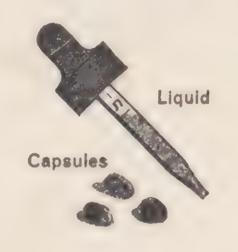
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(1) Wilson, J. L., and Dickinson, D. G.: J. A. M. A. 158: 261, 1955.





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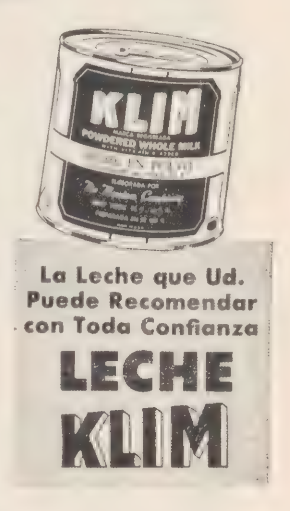
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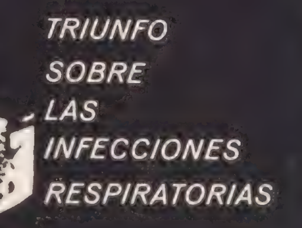
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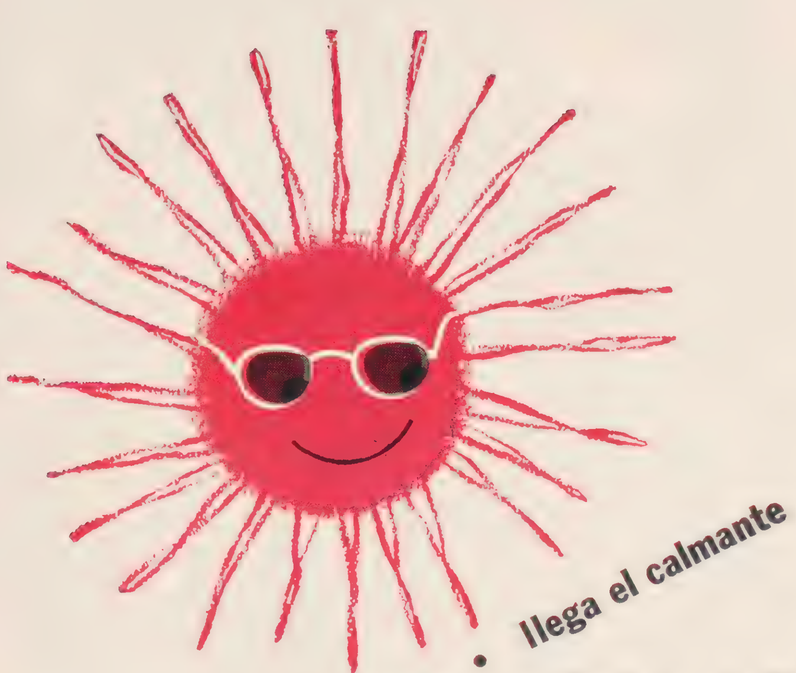
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DE LA ASOCIACION MEDICA DE PUERTO RICO

VOL. 50 MAYO, 1958 No. 5

CHRONIC ADRENOCORTICAL INSUFFICIENCY (ADDISON'S DISEASE):

CASE REPORT*

*AGUSTIN M. DE ANDINO JR., M.D.***
and
JORGE PEREZ CRUET, M.D.

It is remarkable that in Puerto Rico, where tuberculosis has a great morbidity, chronic adrenocortical insufficiency does not appear to be a frequent disease. Upon reviewing our literature this appears to be the first proven case of chronic adrenocortical insufficiency reported.

Although previously the disease had been attributed to tuberculosis in the great majority of instances, it is now known that over 50% of the cases are of idiopathic etiology, the remaining cases being due to partial or complete destruction of the adrenals by tuberculosis, rarely by bilateral tumor metastases, leukemic infiltration, amyloid disease, hemochromatosis or histoplasmosis.

In 1855 Thomas Addison[1] first clearly described the clinical syndrome which he named "Disease of the Supra-renal Capsule", but it was Trousseau[2] who first proposed to call the supra-renal syndrome, "Addison's disease." Addison demonstrated the presence of disease of the adrenal glands in patients with weakness, anorexia, fatigue, anemia, feeble heart action, hypotension, hyperpigmentation, and gastrointestinal irritability. Proof of the vital function of the adrenals was demonstrated by Brown-Sequard[3] in 1856 by the experimental production of an exaggerated Addison's disease by adrenalectomy.

CASE PRESENTATION

A 48-year old mulatto housewife was seen for the first time at the Out-Patient Department of the San Juan City Hospital on

---

* Paper submitted by Dr. Pérez Cruet to the Committee on Awards of the School of Medicine, University of Puerto Rico, in fulfillment of the requirements for the Dr. R. Ruiz Arnau Memorial Prize in Medicine, 1957.

** From the Department of Medicine, School of Medicine, University of Puerto Rico and the Department of Medicine and the Endocrine Clinic San Juan City Hospital, San Juan, Puerto Rico.

February 21, 1956 with the chief complaint of generalized weakness, nausea, anorexia, and epigastric distress of about eight months duration. She complained of night sweats but denied any chest pain, respiratory distress or bloody sputum. She had been fairly well until about eight months previously when she noticed occasional epigastric distress and generalized malaise which alternated with periods of normalcy. This was accompanied by loss of appetite, generalized weakness, and weight loss. She denied any history of bulky stools, diarrhea, or low abdominal pain. The above symptoms persisted and by December 1955 the epigastric distress became worse. In January 1956 she was taken to a private physician because of epigastric distress, anorexia, generalized weakness, weight loss, fever, and chills. A diagnosis of "general debility" was established for which the patient received several injections and pills. She improved but continued to have the above symptomatology and hospitalization at the Gurabo Municipal Hospital was necessary. On admission the blood pressure could not be taken. Intravenous fluids were given with some improvement and she was discharged two days later. She continued to have epigastric distress, nausea, weight loss, generalized weakness, and vomiting that aggravated her condition.

The patient denied any history of jaundice, hematemesis, hemoptysis, or melena, but admitted constipation. She had pneumonia in 1932. During the previous year her sister had noticed that the patient's skin was becoming darker. There was a total weight loss of about 20 pounds. Twenty years previously she had been told to have pernicious anemia. A history of pulmonary tuberculosis in a cousin was obtained.

The menarche was established at the age of 13 years. The last menstrual period was in December 18, 1955. Patient was a grava VI, paragravida VI, abortion O. The diet had been grossly deficient consisting of: Breakfast: milk. Lunch: orange juice and consomme. Supper: usually nothing.

The psychiatric history revealed that the patient was worried by minor problems. She showed a suspicious attitude towards her surroundings. With the onset of her illness she had become more irritable and apprehensive.

On physical examination the blood pressure was 60 ? and the pulse was very feeble and practically absent. Respiratory rate was 18 per minute. Temperature: 98.2 F. The patient was a well developed, undernourished mulatto female who appeared chronically ill.She could not sit or stand by herself because of general weakness. Her voice was of low pitch and she talked slowly. The eyes were sunken and soft. Pupils reacted to light and accommodation. Small patches of dark brown pigmentation were seen on the oral mu-

cosa. The breasts were pendulous and relatively large and showed hyperpigmented areola and nipples. Occasional coarse inspiratory râles were noted on both bases which disappeared after deep inspiration and cough. The heart was small to percussion. The heart rate was 90 per minute and the heart sounds were distant and faint. Examination of the genitalia revealed a black macular area two cm. in diameter on the left labia minora. No abnormalities of the cervix were present. The extensor surface of the skin of the upper extremity was hyperpigmented especially at the knuckles and the elbow joints. Scattered dark brown freckles were observed in the palms of both hands. A hyperpigmented area around a scar on the left leg was observed. No papular or skin lesions were observed. The clinical impression was chronic primary adrenocortical insufficiency complicated by pulmonary tuberculosis.

COURSE IN THE HOSPITAL

Upon admission on February 21, 1956 she was given 200 mgm. Cortisone, 10 mgm. Cortate, and 1,000 c.c. of 5% glucose in saline. After this treatment was started the blood pressure increased to 96/60. She was afebrile.

A regular diet with 20 grams of carbohydrate snacks between feedings was ordered.

A maintenance regime of 25 mgm. of Cortisone, 4 mgm. of D.O.C.A. (Cortate), and 8 gm. of NaCl was prescribed. Antituberculous treatment with 300 mgm. of Isoniazid and 1 gram of streptomycin daily was started on 3-2-56.

On 3-1-56 the skin pigmentation had practically disappeared. The patient was afebrile and doing well.

Under the above therapy the patient's course in the hospital was essentially uneventful. She was free of previous symptomatology except for continuous cough and expectoration of whitish sputum.

The maintenance therapy was interrupted on 3-27-56 to perform special metabolic studies. The patient started to develop painful nodules at the site of previous injections and to complain of general malaise and loss of appetite on 4-4-56.

On 4-7-56 she developed an adrenal crisis characterized by abdominal distress, extreme weakness, tingling sensation in the extremities, and restlessness. She refused meals. At that time 1,000 cc. of normal saline solution was given intravenously after which the patient improved. The blood pressure was very difficult to detect. The same day she started to vomit and to refuse meals.

Maintenance therapy was restored on 4-17-56 because of impending adrenal crisis. On 4-18-56 she developed serious pyrosis

and severe epigastric pain. Two days later she developed psychotic manifestations and started to talk nonsense. The maintenance therapy was continued but from 4-24 to 4-27 cortisone was temporarily discontinued. Psychotic manifestations disappeared with the last measure. 12.5 mgm. of cortisone were started with no observable psychic disturbances on 4-28-56. The patient was essentially asymptomatic thereafter.

On 6-6-56 the patient was transferred to the Ruiz Soler Sanatorium for further antituberculous treatment.

It is important to mention that on admission the patient weighed 102 pounds. When she was transferred to the Ruiz Soler Sanatorium her weight was 128 pounds.

LABORATORY DATA AND SPECIAL STUDIES:

1. Complete blood count: (C.B.C.):
   2-21-56 - R.B.C. 4,420,000; W.B.C. 8,500; Hb.; 13.2 Gm.
   2-22-56 - W.B.C. 8,400; Hb.; 13.0 Gm.
   2-23-56 - R.B.C. 5,000,000; W.B.C. 7,700; Hb.; 13.2 Gm.
   3-1 -56 - Hb.; 13.0 Gm.
   3-19-56 - R.B.C. 4,190,000; W.B.C. 9,960; Hb.; 12.0 Gm.
   4-3-56 - Hb.; 15.3 Gm.
   4-4-56 - Schilling differential: Baso. 3%, Eo. 8%, myelo. 3%, juvenile-stabs 2%, Seg. 36%, Lymph. 23%, and Monos. 25%.

2. Urinalysis:
   2-22-56 - Sp. gr. 1.010; alb.: neg.; S.: neg.; Sediment: w.b.c. 12 to 15 hpf; r.b.c.: 0 to 2 hpf; epith. cells: 0 to 3 hpf; granular and hyaline casts.
   2-24-56 - Turbid specimen Alb.: neg.; S.: neg.; Sediment: wbc: 10 to 20/hpf.; rbc: 1 to 3/hpf.
   2-27-55 - Sp. gr.: 1.010; wbc 2 to 5/hpf.; rbc.: 3 to 5/hpf.
   2-28-56 - Sp. gr.: 1.030; wbc 0 to 2/hpf.; rbc.: 0 to 4/hpf. alb. neg; Sugar: neg.

3. Feces:
   2-22-56 - Feces for ova and parasites: negative.
   2-24-56 - " " " " " " .
   3-13-56 - " " " " " " . No occult blood.

4. Sodium determination in the serum:
   4-3-56 - Serum sodium: 131.0 mEq. L or 301.0 mgm. %.

5. Potassium determination in the serum:
   4-3-56 - Serum potassium: 5.55 mEq. L or 21.7 mgm. %.

6. Chloride determination in the serum:
   2-23-56 - Serum chloride: 119 mEq./L
   2-27-56 - Serum chloride: 109 mEq./L

7. Fasting blood sugar:
   2-21-56 - F.B.S.: 55.3 mgm. % (on admission).
   2-27-56 - F.B.S.: 78.8 mgm. %.
8. Blood urea nitrogen:
   2-23-56 - B.U.N.: 25.8 mgm. %.
9. Sedimentation rate:
   2-23-56 - Volume of packed cells: 46 mm.
   3-19-56 - Sed. rate 38 mm/hr.
10. CO^2 combining power:
   2-23-56 - 25 mEq./L
11. Smear of sputum and culture for Mycobacterium tuberculosis:
   3-3-56 - Smear: negative; No acid fast growth in culture media.
   3-7-56 - Smear: neg.; No colony growth in 2 weeks.
   4-10-56 - Smear: neg.; Culture for Mycobacterium tuberculosis: No growth.
12. 17-ketosteroids in the urine:
   2-23-56 - 8.12 mgm. per 24 hrs. (under cortisone therapy)
   4- 5 -56 - 3.87 mgm. per 24 hrs. (after cortisone was discontinued).
13. Thorn test:
   4-6-56 - First sample: 516; Second sample: hemolyzed.
   4-10-56 - First sample: 840; Second sample: 600 (28% drops).
14. Glucose tolerance test:

4-2-56 -	1st	(F)	71.4 mgm./100 c.c. of blood
	2nd	½ hr.	114.2 mgm./100 c.c.
	3rd	1 hr.	117.8 mgm./100 c.c.
	4th	2 hrs.	132.1 mgm./100 c.c.
	5th	3 hrs.	96.4 mgm./100 c.c.
	6th	4 hrs.	52.0 mgm./100 c.c.
	7th	5 hrs.	71.4 mgm./100 c.c.

15. Oral Kepler test (Positive test)

4-6-56 -	10:00 to 7:00	750 c.c.
	7:00 to 8:00	110 c.c.
	8:00 to 9:00	80 c.c.
	9:00 to 10:00	200 c.c.
	10:00 to 11:00	50 c.c.

An electrocardiogram taken on 2-23-56 revealed a normal sinus rhythm with an auricular and ventricular rate of 100; P. R. interval 0.16; QRS interval 0.10; QT interval 0.32; electrical axis was + 25. There was low voltage throughout.

16. Roentgenologic studies:

(a) 2-21-56. Chest X-ray: The heart is very small in the

transverse diameter with a cardio-thoracic ratio of 7.5 to 25 cm. There is a fine nodular infiltration through the right lung field more accentuated at the level of the second anterior interspace. In the left side there is a minimal fibroid infiltration in the left intraclavicular region.

(b) 2-24-56. Scout film of the abdomen shows no calcification of the adrenals and is essentially negative.

(c) 3-10-56. Reexamination of the chest demonstrated an increase in the transverse diameter of the heart when compared with previous films of 2-21-56. The pulmonary changes previously described are slightly less conspicuous but persistent in the right upper lobe.

(d) 3-16-56. Reexamination demonstrated no significant change when compared with previous films.

(e) 5-2-56. Reexamination of the chest demonstrated that the originally described pulmonary findings are less prominent.

(f) 5-21-56. When compared with examination on 5-2-56 there is no significant change in the fibroid infiltration throughout both lung fields. The heart is within normal limits in size and shape, and there is some calcification of the arch of the aorta.

DISCUSSION

In the original description of this disease Addison[1] demonstrated the presense of abnormality of the adrenal glands in patients with weakness, anorexia, fatigue, anemia, feeble heart action, hyperpigmentation, gastrointestinal irritability, unequivocal signs of feeble circulation, and general prostration. In addition, these patients usually manifest weight loss, dehydration, hypotension, nausea, dizziness or syncopal attacks, salt craving, muscle pain, mental symptoms, changes in the gonadal function and secondary sex characteristics, and hypoglycemic manifestations such as sweating, trembling, loss of initiative, unconsciousness, dilated pupils, desorientation and blurring of vision.

The usual laboratory findings in this condition consist of a decreased serum sodium and chloride concentration, small heart size (chest X-ray), low urinary 17-ketosteroid and corticoid excretion, flat oral glucose tolerance curve, hemoconcentration, abnormal electroencephalogram, X-ray demonstration of the presence of healed and or active tuberculosis in many instances, increase in circulating lymphocytes with normal to high levels of circulating eosinophils, and electrocardiographic changes suggestive of hyperkalemia.

In the differential diagnosis of this disease the most important and prominent feature to consider is hyperpigmentation which

is one of the cardinal signs of Addison's disease. However, we must mention that Lewin[4] has listed 64 proven cases without pigmentation. Vallejo[5] also reported adrenal insufficiency without melanoderma. In our milieu, we should remember that patients of Negroid extraction normally may show increased pigmentation indistinguishable from that occurring in patients with Addison's disease and that occasionally, vitiligo is the only evidence of disturbance in pigmentation.

Among other diseases to be mentioned and considered in the differential diagnosis we should mention tropical sprue, chronic infections such as tuberculosis and brucellosis, hypopituitarism of pregnancy, panhypopituitarism, malnutrition, neurasthenia, thyrotoxic myopathy, myxedema, hemochromatosis, heavy metal poisoning, wasting disease such as intra-abdominal malignancy with skin pigmentation (acanthosis nigricans), chloasma, spontaneous hypoglycemia, renal insufficiency of the "salt-loosing type", hyperparathyroidism, and porphyria.

At the present time it is known that chronic primary adrenal insufficiency may be congenital or acquired, complete or partial. The former subdivision in many instances fails to be recognized[6]. In many cases classified as of unknown etiology a Mendelian trait has been incriminated. The congenital type is encountered, as a rule, in infants who often die in the neonatal period and whose mothers supposedly had been deficient in adrenal cortical secretions.[7,8]

Injudicious use of steroid therapy for prolonged periods of time may produce a partial or complete depression of the adrenal cortex, usually of a temporary nature.

A discussion of the influence of the adrenal on the stomach, brain, heart, skin and other organs is beyond the scope of this paper. Gray et al[9] have studied recently the adrenal influences on the stomach and particularly the occurrence of peptic ulcer in Addison's disease during adrenal steroid therapy. They suggest that antacid therapy may be indicated in patients with Addison's disease maintained on a long-term corticoid therapy. On reviewing the literature the occurrence of chronic peptic ulcer disease in patients with Addison's disease is found to be quite rare.

Sanford and Favour[10] have recently studied the interrelationship between Addison's disease and active tuberculosis. In a review of 125 cases they found that 24% of the cases had a clinical history of tuberculosis. They concluded that active tuberculosis in the presence of adrenal insufficiency shows a tendency toward chronicity and relapse similar to the patients with tuberculosis alone. They also concluded that in a patient with known adrenal insufficiency the appearance of increased symptoms, increased

hormonal requirements, tachycardia, fever, leukocytosis with a relative lymphopenia, and an elevated sedimentation rate should suggest the presence of either active tuberculosis or some similar chronic infection.

SUMMARY AND CONCLUSIONS

1. The first case report of proven chronic adrenocortical insufficiency (Addison's disease) in Puerto Rico is presented.

2. The diagnosis of Addison's disease must be entertained in any patient with hypotension, hyperpigmentation, ill-defined gastrointestinal complaints, rapid weight loss, and intercibal or fasting attacks of dizziness or loss of consciousness.

3. Prompt diagnosis, intelligent management and proper cooperation from the patient are essential for successful treatment of this disease.

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PNEUMOCONIOSIS DE BAGAZOSIS, CEMENTO Y MAGUEY*

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Por la importancia que tiene en el azúcar de caña, me ha sido encargada la presente comunicación, que tratará de los daños que a la salud ocasiona el polvillo de caña derivado del bagazo, material residuo que queda en la caña después de la extracción del azúcar. En la mayoría de las factorías de azúcar se usa el bagazo seco y trozado, por sus cualidades madereras, como combustible de las calderas de cobre en las que se verifica la concentración del azúcar. De esta manera, el bagazo es aprovechado de inmediato y en el sitio mismo de la faena.

Debido a sus cualidades aislantes y a la resistencia de sus fibras, el bagazo también se presta para la fabricación de cartelones livianos en edificaciones y como material aislante contra ruídos y temperatura, y es así como, bajo el nombre comercial de Celotex que se le conoce en Norte América.

La inhalación de polvillo de bagazo puede originar la enfermedad pulmonar llamada bagazosis o fiebre de bagazo. Fué observada por primera vez en 1940, en el estado de Louisiana, en un joven negro estibador de Nueva Orleans que descargaba fardos de bagazo en una atmósfera rica en polvillo y quien enfermó en forma aguda, febril, con disnea, tos y desgarro hemoptoico. En el tórax se escuchaban estertores subcrepitantes y la radiografía señalaba un sombreado miliar sobre ambos pulmones, el que había desaparecido en un control verificado a los dos meses. Los norteamericanos JAMISON y HOPKINS publicaron este caso en 1941. El año 1942 publican los ingleses CASTLEDEN y HAMILTON 4 casos más de bagazosis, entre los cuales hubo uno fatal.

Hasta el comienzo de la Segunda Guerra Mundial se transportaba el bagazo en su estado natural de humedad, siendo ablandado y trozado en grandes tinas. Pero, a fines del año 1939, con el fin de ahorrar espacio en los barcos de carga, casi se llegaba a prensar los fardos de bagazo y, como ya no era posible un ablandamiento, eran partidos con picota y sus trozos lanzados a una máquina trituradora que completaba la faena, originándose nubes de polvillo. Los obreros trabajaban por turnos diurnos y nocturnos que se sucedían cada dos semanas. En la noche se obscurecían las ventanas por el peligro de los bombardeos; no existía sistema purificador del aire. De esta manera enfermaron en Ingla-

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terra de bagazosis, entre el invierno de 1939 y la primavera del 1941, de 21 obreros, 10 de ellos, es decir el 47.5%. La enfermedad se hacía presente, por término medio, después de 8 semanas de exposición en la faena. Se comprobaba alta temperatura, frecuentemente disnea marcada, tos violenta con escaso desgarro; en 4 casos, hemoptisis. A menudo, se constataron sudoraciones y pérdida de peso.

El inicio de la enfermedad puede ser extraordinariamente repentino. RAVINA menciona un ciclista que, en estado de perfecta salud fué atacado por tan brutal acceso de tos que a duras penas pudo alcanzar su vivienda situada a veinte metros. También cita el caso de un obrero que en mitad de su jornada sufre grandísima opresión torácica y tos ininterrumpida.

La fiebre se mantiene entre dos y seis semanas bajo la apariencia de una bronquiolitis aguda o de una neumonía atípica. En el tórax se escuchan estertores de gruesas burbujas. Cuando existe la enfermedad hay cuadros radiográficos positivos, los más se esclarecen en forma de un sombreado miliar que se tupe y muestra tendencia a confluir hacia los hilios. Pasadas 6 a 8 semanas, acostumbran a despejarse radiológicamente los pulmones, hasta normalizarse completamente.

Tres años más tarde, HUNTER y PERRY examinaban a estos enfermos ingleses y no comprobaban nada patológico en las radiografías, pero algunos de ellos aún se quejaban de disnea y tos. Fuera de estas molestias residuales prolongadas, existen casos crónicos que llegan a la muerte después de largo padecimiento. Como ejemplo, sirva un electromecánico del grupo de los obreros ingleses, que montó y reparó la máquina cortadora en el local de la faena del bagazo, por espacio de 13 meses. También él enfermó en septiembre de 1940 con las manifestaciones corrientes de una bronquitis y falleció, después de repetidas hospitalizaciones en octubre de 1944. La autopsia mostró una bronquitis supurada y una traqueítis, una fibrosis pulmonar y una bronquiectasia. También se halló fibrosis pulmonar en la necropsia de un caso fatal de una serie de 12 enfermos de bagazosis, publicado en Louisiana por SODEMANN y PULLEN. Resultados similares comunicó el anatomopatólogo SALFELDER, de Mérida, Venezuela, en el año 1956.

El factor o los factores etiológicos que condicionan la aparición de la bagazosis no se han aclarado aún lo suficiente. Los norteamericanos SODEMANN y PULLEN han encontrado por medio de la biopsia practicada en la sexta (6) y séptima (7) semana de evolución, como también, en la autopsia, una reacción fibroblástica en el tejido intersticial del pulmón y agujas ópticamente activas que semejan el polvillo de bagazo. Admiten que estas par-

tículas de polvillo originen la reacción pulmonar y creen que son desintegradas, a la larga, o eliminadas por absorción, lo que explicaría la normalización del cuadro radiográfico.

De otra opinión son LEMONE, SCOTT, MOORE y KOVEN quienes ven en el contenido de ácido silícico del bagazo el origen de la enfermedad. El polvillo encontrado en las fábricas de bagazo contiene un 1% de proteínas y se compone principalmente de fibras de celulosa, aunque una porción de ellas se encuentre en forma cristalizada. Sometido a 400 grados de temperatura, se obtiene del bagazo 3% de ceniza, compuesta en su mitad de ácido silícico amorfo. El residuo contiene cuarzo de gránulos de un diámetro de 20 a 30 micros. que, desde el punto de vista microscópico y rontgenespectroscópico hacen un 3 a un 5% del total de la ceniza, o sea, 0.1 a 0.2% del bagazo, según cálculos del investigador británico NAGELSCHMIDT.

En el bagazo que llevé desde Venezuela en 1955, con motivo del Tercer Congreso Panamericano, se encontró 2.9% de ácido silícico. Atendiendo al diámetro de los corpúsculos de 20 a 30 micros. y al bajo contenido de ácido silícico es muy reducido el riesgo de contraer silicosis en la faena del bagazo. Yo quisiera también desechar esta teoría, en atención a que la eclosión febril aguda de un comienzo y el restablecimiento y normalización ulterior del cuadro radiográfico no se avienen con la evolución de una silicosis.

Un tercer punto de vista representan los ingleses CASTLEDEN y HAMILTON-PATERSON, quienes ven en la bagazosis una enfermedad alérgica. Fundamentan su opinión en una reacción cutánea a base de extracto de bagazo obtenida positiva en tres enfermos de un grupo de cuatro. Esta reacción intradérmica no ha sido confirmada por investigadores posteriormente. Tampoco se ha observado eosinofilia en el hemograma de estos enfermos.

Es así como llego yo a una cuarta ponencia, en la que juegan importante papel en la constitución de la enfermedad los microorganismos que viven en el bagazo. En este sentido, es muy interesante el contenido de un 4% de glucosa en los fardos de bagazo comprobado en Louisiana y en la falta de azúcar constatada a su arrivo en Inglaterra, azúcar que habría sido fermentada manifiestamente por hongos. DUNCAN pudo observar en un gramo de bagazo obtenido del local de faenas de corte mecanizado (shredder), 240 millones de esporas de hongos, en su mayoría, de la cepa Puccinalis. En el cultivo, se pudo aislar alrededor de 20 distintos hongos, entre ellos: Paelomices varioti, Aspergillus fumigatus, Aspergillus niger, Aspergillus terreus, Aspergillus candida, Trichoderma lignorum, Monilia sitophilia, Aleurisma y especies Pennicillium, Mucor y Rhizopus. El papel que juegan estos hongos en

la enfermedad es puesto de manifiesto en experimentación animal por GERST, TAGER y MARINARO de Chicago. Aplicaron a conejos polvillo fresco logrando obtener en pulmón y órganos parenquimatosos focos de necrosis, y fuerte reacción celular, con la presencia de hongos o esporas. Con polvo esterilizado solo alcanzaron reacción a cuerpo extraño. Estas diferencias en la calidad de reacción la basan en la influencia de los hongos. Les fué posible aislar por el cultivo diferentes microorganismos del pulmón, entre ellos el Aspergillus fumigatus. Repitieron las experiencias con masiva incorporación de hongos, con lo que se demostró el Aspergillus fumigatus como el más peligroso y activo formador de focos necróticos.

Conjuntamente con mi colaborador de muchos años, el Prof. EHRHARDT, actual director del Instituto de Medicina del Trabajo en Jena, hemos llevado, después de nuestra común visita a Mérida, Venezuela, trozos de bagazo a Alemania, de los cuales se pudo aislar 8 cepas de hongos: cuatro específicas de Aspergillus, dos especies de Paecilomyces y dos especies de Rhizopus. Todas estas cepas proliferaban a 37 grados mejor que a 25 grados, hecho especialmente notorio en el Aspergillus fumigatus y Rhizopus ozyzae.

Los gráficos que acompañan los trabajos experimentales de los norteamericanos antes citados, no dejan ver con claridad las alteraciones patológicas del pulmón. Es por ello que repetimos la experiencia en Alemania en 10 ratas blancas (GUTHERT, ERHART). Tampoco nos atrevemos aún a entregar una información completa y definitiva sobre la patogenia formal de las alteraciones pulmonares. De todas maneras, podemos ya separar dos procesos fundamentales: de un lado, típicas alteraciones inflamatorias y del otro, reacciones a cuerpo extraño originadas por el parénquima pulmonar consecutivamente a la inhalación de las partículas de polvo. Las alteraciones inflamatorias son de naturaleza aguda y crónica. Desde una bronquitis aguda supurada, se desarrollan abscesos y cavernas broncogénicas, con bastante contenido en detritus. También se observan pequeños abscesos alveolares diseminados en el pulmón. Las alteraciones crónicas consisten en una destrucción como en enrejado de la pared bronquial y su reemplazo por tejido de granulación, como también en un engrosamiento fibroso parcial del tejido de sostén del pulmón. Es muy probable que estas alteraciones inflamatorias sean producidas por los micelios de los hongos que acompañan al bagazo. Las reacciones a cuerpo extraño se señalaron como nodulillos cabeza de alfiler, de consistencia más dura que los alrededores en la superficie pulmonar o en el corte. Atendiendo a su coloración tienen semejanzas con el polvillo de bagazo.

Así pues, con el resultado de las investigaciones alemanas,

puede decirse que en la bagazosis se trata de una combinación íntima de alteraciones inflamatorias con predominio de lo que llamaríamos reacción a cuerpo extraño del pulmón. De aquí que vimos algunos animales en los que predominaba la reacción a cuerpo extraño y, en cambio, en otros predominaba el componente inflamatorio. Los ensayos deben ser continuados aún.

El pulmón bagazósico pertenece a un grupo de raras neumoconiosis, las originadas por polvos orgánicos. A éstas pertenece también la "Farmerlunge" (pulmón de granjero), y la byssinosis o pulmón del algodonero, con las que tiene analogías en el comportamiento clínico y morfológico. También en la fiebre del lino y de la del cáñamo (cannabosis) se conocen manifestaciones parecidas. La "Farmerlunge" se ha observado mucho en el último decenio en los E.U. y en la isla inglesa de Jersey en el Canal de la Mancha y en Gran Bretaña misma. Se presenta epidémicamente después de veranos lluviosos entre jornaleros que han trabajado con heno o trigo enmohecido. El signo jefe de la enfermedad es una disnea progresiva, conjuntamente con fiebre y pulso alto, tos con escaso desgarro consistente. Clínicamente se comprueba en el pulmón ruidos de catarro o asma bronquial. En los casos serios, la disnea se hace amenazadora. Se llega a hemóptisis y a la expulsión de un desgarro mucopurulento, en el que se detectan hongos. Este desgarro huele como a la levadura de cerveza. Como causantes de la "Farmerlunge" fueron señalados los hongos del moho: Aspergillus, penicillium y mucor, los que también se encontraron siempre en el desgarro de obreros cosechadores, enfardadores de heno y granjeros. Se trata, pues, aquí de una micosis pulmonar profesional. De ésta se diferencia la bagazosis, en la que sólo ocasionalmente se comprueban hongos en el desgarro, como fué en el caso crónico y fatal, cuyo desgarro contenía Aspergillus, el cual no fué encontrado en el pulmón autopsiado.

Frecuentemente se tomaba el "Farmerlunge" o "pulmón de granjero" por una tuberculosis, hasta que tras reiterada y continuada ausencia de bacilos, se llegaba a encontrar la causa en los hongos. También la bagazosis se toma con no rara frecuencia por una tuberculosis pulmonar.

Las enfermedades pulmonares ocasionadas por el algodón son originadas por el polvo de la fibra y de su semilla durante la cosecha, la separación de fibra y semilla, la enfardadura, la selección, la mezcla, el peinado y alisamiento, el lustre, el hilado y el tejido. Sobre todo ésto han aparecido numerosos tratamientos desde hace cien años. CAMINITA y colaboradores publicaron en Washington en 1947 en la Editorial Nacional de los Estados Unidos una síntesis bibliográfica de 247 publicaciones.

Los anglosajones diferencian 4 enfermedades distintas de las

vías respiratorias: La "fiebre de las hilanderías" que asalta frecuentemente a los novicios de las hilanderías de lino, cáñamo, yute o algodón y que desaparece sin tratamiento en dos o tres días. La "tos de los tejedores" que aparece sólo ahí donde se trabaja con hilo enmohecido; la bisinosis o "pulmón de algodonero", una bronquitis crónica que puede llevar a la muerte y que, en Gran Bretaña, Francia y algunos estados norteamericanos es considerada enfermedad profesional con indemnización obligatoria. Estima COLLIS que el 86% de los obreros ingleses mayores de 20 años que trabajan el cardado en las hilanderías sufre de bisinosis que tiende a agravarse regularmente. La cuarta enfermedad se presenta sólo en trabajos con algodón coloreado de bajo rendimiento y se le llama con el nombre de su investigador, "enfermedad de Neal". Transcurre en tres o cuatro días, con fiebre de 39.3 a 39.7 grados y escalofríos. También la he observado en Alemania (Westfalia).

NEAL y SCHEITER lograron aislar del algodón un organismo gram-negativo, bacilar, que no forma esporas, el Aerobacter cloacae y achacan a la inhalación de su endotoxina la causa de la enfermedad como, también, al Aerobacter cloacae y su toxina, el origen de la bagazosis. Contra lo arriba expuesto, debe decirse que esta hipótesis no ha sido comprobada en absoluto y que el curso clínico y rontgenológico de la bagazosis difiere fundamentalmente de la corta enfermedad de NEAL.

Si se me permitiera hacer un cierto paralelo entre la bagazosis y otra enfermedad, llamaría la atención sobre la suberosis, o enfermedad por aspiración de polvillo de corcho, la que es estudiada por el Prof. JORGE DE SILVA HORTA y el especialista en broncopulmonares LOPO DE CARVALHO CANCELA, ambos de Lisboa, Portugal, país que aporta, con sus 977 factorías, más o menos la mitad de la producción mundial del corcho.

La enfermedad ha sido observada, hasta el momento, en los Estados Unidos (JAMISON, SODEMAN, y otros), Inglaterra (CASTLEDEN, HUNTER y otros), Italia (GUARIGIONE), México (MANAS), Venezuela (SALFELDER). El que ella cobre importancia en la República Dominicana, no estoy por ahora, facultado para afirmarlo. De todas maneras, parece importante que sus médicos conozcan el cuadro clínico y, ante enfermedades similares, piensen en la bagazosis.

Una terapia específica aún no se conoce, lo que es explicable dada la inseguridad de su etiología. Las sulfonamidas se han demostrado como ineficaces.

Como profilaxis, se aconseja en todas partes la protección con-

tra el polvillo de bagazo. Son interesantes las indicaciones de MIDDLETON, del Servicio Médico del Trabajo Inglés, quien consiguió en la primavera de 1941, erradicar la presentación de nuevos casos durante los primeros años de la Segunda Guerra Mundial. Para ello se valió de instalaciones absorbedoras de polvo en los locales de faena como, también, del tratamiento por medio de un chorro de agua dirigido sobre la sierra cortadora, lo que tenía por objeto humedecer las fibras del fardo de bagazo. Sólo en 1944 se vinieron a observar casos aislados de la enfermedad.

Como se me exteriorizara el deseo de que hablara en esta ocasión sobre los efectos del polvillo de cemento, abordaré este tema a continuación. Los cementos son mezclas ricas en calcio y silicatos que, al agregarles escasa cantidad de agua, originan una sustancia petrosa que se aprovecha para construcciones. Hay diferentes clases de cemento: el de Portland, el cemento Hutte, el cemento bauxita (ciment fondu). El cemento Portland, conocido desde hace 135 años se compone de piedra caliza que se ha incinerado en hornos giratorios a 1500 grados C. en unión de greda y marga. Este producto de calcinación, al cual se agrega algo de yeso, se tritura finalmente en molinos de ruedas, lo que origina una intensa polvareda. El cemento Hutte ocupa, en vez de greda o marga, escoria de altos hornos y es fabricado por los grandes estados industriales europeos: Alemania, Francia, Inglaterra. El "ciment fondu" es confeccionado por estados poseedores de yacimientos de bauxita. Pero, para los fines de nuestras observaciones desde el punto de vista de la medicina del trabajo, ésto carece de importancia, ya que toda fabricación de cemento origina gran cantidad de polvillo, el que se compone de alrededor de un 60% de óxido de calcio y un 20% de dióxido de silicio, pero sin presentar cal ni ácido silícico libre, sino combinado en forma de aluminato y silicato. Suele encontrarse ácido silícico libre hasta en una proporción de un 6%. La mayor génesis de polvillo la encontramos en la trituración de la materia prima, en la sala de depósito, empaquetadura o ensaque y en el transporte. En las fábricas modernas ha disminuído, mediante la automatización, el peligro del polvillo. Los higienistas admiten un contenido máximo de 50 mg. de polvillo por centímetro cúbico de aire.

El polvillo de cemento puede originar muy diversas enfermedades en la piel y mucosas, en pulmones y estómago. Las lesiones cutáneas del cemento revisten la forma de abscesos y cauterizaciones que se achacan a la acción directa de las partículas calcáreas sobre la piel. Demuestran su extraordinaria frecuencia las expresiones populares francesas de "pigeonaux, rosignols, perdrix". Los llamados arañazos del cemento (un eczema) se encuentra naturalmente más a menudo entre albañiles y concreteros, en los

que puede observarse su crecimiento desde pequeños nódulos pruriginosos que infiltran e induran la piel, y que pueden llevar a la piodermitis por infección agregada.

Las alteraciones de las mucosas se manifiestan como inflamaciones agudas o crónicas de las conjuntivas, de la mucosa bucal y nasal, con enrojecimiento, edema, secreción y, tardíamente, atrofia y formación de úlceras y hemorragias. No es infrecuente encontrar úlceras profundas con perforación del septum nasal.

Por aspiración del polvillo del cemento en las cavidades nasales se originan concrementos sólidos llamados rinolitos (Nasensteine). También las mucosas de las vías aéreas superiores sufren irritaciones que se traducen por catarros agudos y crónicos, con tos y expectoración. Investigaciones seriadas de cientos de trabajadores del cemento en Alemania (Mehle), Francia (Jullien), Italia (Antoniotti) señalan a cada cuarto obrero, padeciendo de tos y expectoración y a un 3 a 4% de bronquitis, En los obreros con más de 10 años de actividad se encontró 7 a un 9% de bronquitis. El francés Andre Feil (de la medicina del trabajo) constató irritaciones de las vías aéreas superiores en un tercio de mil obreros del cemento.

Respecto a los cuadros radiográficos, no hay unanimidad de opiniones entre los investigadores. Los norteamericanos GARDNER, SAMPSON, VORWALD creen que el polvillo de cemento sea inocuo para los pulmones. Los italianos CACCURI, AUDOGIANOTTI y PRISCO describen una fibrosis pulmonar que se exterioriza en las radiografías como un ensanchamiento en forma de salchicha de los hilios (forma hiliar) o como un sombreado finísimo difuso y homogéneo (forma pulmonar). Estas neumoconiosis son raras y sólo se observan después de decenios trabajados en el cemento. Puedo describirles un caso de éstos particularmente grave que mostraba una radiografía como de silicosis en tercer estadio. Este enfermo había trabajado 26 años en el horno de una fábrica de cemento alemana, en el ensaque y en el depósito. A los 36 años se le tomó la radiografía que muestro en este cuadro. Se agravó progresivamente; a los 44 años era ya un inválido y a los 47 moría en Heidelberg. La autopsia, hecha por el anatomopatólogo Prof. DOOR, demostró en ambos pulmones la existencia de dos cayosidades gris-cemento, del tamaño de un puño de hombre. Histológicamente se evidenciaban numerosos nódulos silicoticoideos, que sin embargo, se diferenciaban claramente de la silicosis por su tejido cicatricial poco denso. También se veían depósitos particularmente ricos de polvillo en el interior de las cicatrices, entre los ralos nódulos y los numerosos granulomas pulverulentos. Esta autopsia enriqueció fundamentalmente nuestros conocimientos sobre la neumoconiosis del cemento, a la

cual DORR designa como silicatosis, mientras otros la quieren incluir entre las silicosis mixtas.

Otros casos graves de la enfermedad han sido descritos por GAUBATZ en Alemania y por SCHIOTZ en Noruega. Por todo lo expuesto, queda fuera de dudas que el polvillo de cemento no sea inocuo para los pulmones, aunque las enfermedades mortales sean muy raras y sólo se produzcan después de exposición prolongada en faena de decenios. Los franceses JULIEN, LEANDRI y BOUVEYRON indican además que el cemento, fuera de originar daños en la piel y órganos respiratorios, es capaz de producir úlceras en el estómago y duodeno. También MEHLE, quien examinó recientemente obreros del cemento en el territorio alemán del Saar, encontró entre ellos un 17% de enfermos del estómago, de los cuales, un 10% con úlceras gástricas. La mayoría de los ulcerosos (79%) había contraído su enfermedad dentro de los 5 primeros años de trabajo. Si consideramos que la población alema total tiene una incidencia en úlcera gastroduodenal de un 2.9 a un 3.2%, se encuentra la cifra de un 10% en las fábricas de cemento, bastante elevada. También es interesante señalar la extrema rareza de la TBC entre los obreros del cemento, de tal manera que ya se ha planteado la teoría que considera al polvillo de cemento como una protección contra la TBC, lo que personalmente considero demasiado arriesgado o aventurado.

Con frecuencia se oyen quejas de la población que vive en las cercanías de las fábricas de cemento, la que es molestada por el fino polvillo, toda vez que éste se distribuye en un área de muchos kilómetros alrededor de la fábrica. Daños en la salud de estos habitantes vecinales no se conocen aún.

Notable puede ser la influencia del polvillo de cemento sobre la vegetación: los norteamericanos PARISH y PIERCE comprobaron una baja en la producción de naranjas y limones en la California meridional, mentras que el alemán EWERT acepta lo contrario en árboles frutales, dado que el polvillo de cemento parece combatir las enfermedades a hongos de los frutales.

Finalmente se me ha pedido que comunique algo acerca del daño pulmonar que puede causar el polvillo de sisal (Maguey). En este punto seré muy breve. En efecto, el trabajo del sisal se desarrolla en un medio de gran densidad en polvo, pero hasta ahora, no ha habido observaciones sistemáticas de ninguna especie acerca de la salud de los obreros del sisal. El francés BOUYEURE designa al polvillo de sisal como apenas dañino y su compatriota WEBER, que observó durante 9 años a 360 obreros de una fábrica de hilo de sisal (ficelle lieuse) no encontró ni neumoconiosis ni manifestaciones alérgicas pulmonares entre ellos. Pero en enero de este año se puede leer un artículo sobre neumoco-

niosis causada por maguey. (British Journal of Industrial Medicine.)

Por último, quisiera reiterarles mi agradecimiento por la paciencia y atención que me han deparado a lo largo de esta disertación, desde la bagazosis, pasando por las enfermedades pulmonares causadas por el algodón, hasta la pneumoconiosis del cemento y el sisal.

OUTBREAK OF FOOD POISONING AT THE SCHOOL OF MEDICINE CAFETERIA TRACED TO GUANABANA SEED (Soursop Seed)

*ROLANDO ARMIJO, M.D.**

This is to report briefly a small outbreak of food poisoning which occurred among medical students and personnel participating of the meal served on the evening of January 31, 1958 and the implications derived from it.

The outbreak

Soon after the supper served on Friday, January 31, several students and personnel in the kitchen evidenced symptoms of nausea, gastric upset and vomiting. By interviewing the participants of that meal, followed by a survey by utilizing a suitable form designed for the purpose, information was collected so as to find out the offending foodstuff, from a total of 33 persons exposed to the suspect meal.

Table I shows the incubation period in eleven cases reported.

TABLE 1: INCUBATION PERIOD

Incubation Period	No. of Cases	Cumulative
Less than 10 minutes	5	5
15 - 45 minutes	3	8
46 minutes - 2 hours	3	11

It can be noted that the great majority of cases were taken ill within a very short period of time after the incriminated meal. This was the first clue which made us think of a chemical intoxication. The disease consisted of nausea, salivation and bad taste in the mouth, gastric discomfort and other symptoms, having a relative frequency as shown in Table 2.

TABLE 2: SYMPTOMS BY FREQUENCY IN ELEVEN CASES

Nausea	11
Salivation and bad taste	9
Gastric discomfort	6
Vomiting	5
Epigastric pain, general malaise and dizziness	2
Mild diarrhea, headache	1

* From the Department of Preventive Medicine and Public Health, School of Medicine, University of P. R.

This disease would not fit into the pattern of any poisonous agent, except that it approaches the clinical picture described for various chemical compounds. In addition to be a rather mild disease, the symptoms subsided in less than one hour in five cases, 1 to 3 hours in five cases and only in one instance, symptoms lasted for 8 hours. All of the patients recovered without any special treatment.

It is believed that a few more students evidenced minor symptoms but did not report them, in addition to these eleven reported cases.

The offending food

It was not necessary to compute attack rates for consumers and non-consumers of each foodstuff, for it became immediately apparent from the survey, that of twelve persons who had drunk guanabana juice, eleven became ill, and of a random sample of twenty-two who evidenced no symptoms, only one had guanabana juice.

This juice had been elaborated in the kitchen from fresh guanabana locally purchased by using a standard machine crushing the flesh and seeds together. A special device separates the smashed seeds and juice fills a receptacle containing ice cubes. Then, the juice was distributed in nearly one hundred glasses and allowed to remain in the refrigerator for about one hour before serving.

It was found that no manipulation nor incubation would have allowed any contamination and growth of enteropathogenic organisms. This was corroborated by negative findings of Dr. Pomales in samples bacteriologically tested. No contact of juice was found to have occurred with metallic receptacles, so as to allow the formation of metallic toxic compounds as described in the past with copper, cadmium or antimonium. The only abnormality found was that seeds had not been removed prior to juice preparation.

Of the nearly one hundred glasses served, it was found that two entire trays containing 70 glasses were left and still kept in the icebox, meaning that only from twenty to thirty glasses had been really consumed. This could account for the low number of cases. Of those who were taken ill it was found that two had had two glasses and in one instance (a cook) probably had five glasses, thus reducing the number of persons exposed to the suspect food to around 16. The most severe case coincided with the one who had probably five glasses of guanabana juice.

In trying to find out any information or evidence about the possible deleterious action of principles contained in the guanabana seed, the following information has been collected.

1.—The Commonwealth Health Department reported that in the past the Government of Venezuela complained that canned guanabana juice processed in Puerto Rico had caused nausea and vomiting among consumers there. The bacteriologic examination of the product proved negative and no further evidence was found.

2.—Dr. Conrado Asenjo, back in 1948, reported in his paper. "The Digestibility of Some Tropical Oils", that "with the only exception of the diet containing guanabana seed oil, the rats consumed all other oil-containing "diets" and furthermore, the summary states that "the animals rejected the guanabana seed oil altogether". It is surprising to find how well the epidemiologic findings among people exposed to juice containing seed remains fit with an observation made ten years ago in the laboratory among rats. Dr. Asenjo says that at the time of the experiment back in 1948 he was puzzled by this rejection of specifically guanabana seed oil.[1,2]

3.—A pilot trial run by Dr. Asenjo with two rats revealed again that both animals rejected a diet containing 20 per cent of guanabana seed oil.

4.—A human feeding experiment with two volunteers proved positive. Both volunteers drank a glass of the suspect guanabana juice, and both evidenced soon after the ingestion, symptoms of nausea, salivation and bad taste in the mouth, belching, subsiding in about thirty minutes.

SUMMARY AND CONCLUSIONS

A food poisoning outbreak involving eleven reported cases is described among consumers of the Cafeteria in the School of Medicine. All the evidence points to guanabana juice improperly prepared, so as to mix the flesh and the seeds together. Past experience and laboratory findings seem to indicate that the guanabana seed may contain some not yet determined chemical principle responsible for the symptoms described in the present outbreak.

Humans seem to be quite susceptible to whatever principle it might be, as eleven persons evidenced symptoms of nearly sixteen who had the guanabana juice, an attack rate of 69 per cent. Those who drank more than one glass of the juice evidenced more marked symptoms and one who drank nearly five glasses was rather severely ill during eight hours.

Since guanabana juice and other products represent rather an important item in Puerto Rican industry, the Department of Biochemistry has demonstrated a special interest in further investigation on this problem, which may lead to the isolation of one

or more substances capable of producing emetic symptoms in man.

The recommendations derived from this finding consisted in elimination of the left over juice and advice to remove the seeds in the future if guanabana juice is served in the Cafeteria.

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MEDIDAS DE PRIMERA AYUDA EN CASOS DE ENVENENAMIENTO*

Traducido de J.A.M.A.—12 de octubre 1957—Vol. 165 (Recommendations of Committee on Toxicology in First Aid Measure for Poisoning.) J.B.D.

El propósito de las medidas de primera ayuda es ayudar a evitar la absorción del veneno. La PRONTITUD es esencial. Las medidas de primera ayuda deben empezarse inmediatamente. Si es posible, una persona debe comenzar el tratamiento mientras otra persona llama un médico. Cuando esto no sea posible, la naturaleza del veneno determinará si se llama primero al médico o se comienza a dar primera ayuda, y luego se notifica al médico. Guarde el recipiente del veneno y algún material sobrante. Si no conoce la procedencia de éste conserve una muestra del vómito.

Medidas a adoptarse antes de llegar el médico

I—Venenos que se han ingerido.

Muchos productos usados en o alrededor del hogar, aunque no se rotulan "veneno" pueden hacer daño si se toman internamente. Por ejemplo, algunas medicaciones, beneficiosas cuando se usan correctamente, pueden causar daño a la vida si se usan impropiamente o en cantidades excesivas.

En todos los casos, excepto los indicados más abajo, REMUEVA EL VENENO DEL ESTOMAGO DEL PACIENTE INMEDIATAMENTE, induciendo el vómito, pues es esencial en el tratamiento, y por lo regular, un medio de salvar la vida. Evite el enfriamiento envolviendo al paciente en frisas si es necesario. No dé alcohol en forma alguna.

No provoque el vómito si:

1. El paciente está en cama o inconsciente.
2. Si el paciente está en convulsiones.
3. Si el paciente ha ingerido productos de petróleo (i.e. "kerosene", gasolina, líquido para encendedores)
4. Si el paciente ha tragado veneno corrosivo (Síntomas: Dolor severo, sensación de ardor en la boca y garganta, vómito).

LLAME AL MEDICO INMEDIATAMENTE:

1. Acido y corrosivos ácido "similares": Sulfato ácido sódico (limpiador de inodoro), ácido acético glacial, ácido sulfúrico, áci-

do nítrico, ácido oxálico, ácido hidrofluórico (removedor de moho), iodo, nitrato de plata (lápiz estíptico).

2. Corrosivos alcalinos: Hidróxido de sodio, lejía (limpiador para tuberías) carbonato sódico (soda para lavado), agua amoniacal, hidroclorato de sodio (substancia descolorante).

Si el paciente puede tragar después de haber ingerido el veneno corrosivo, las siguientes substancias (y cantidades) pueden ser administradas:

Para ácidos: Leche, agua o leche de magnesia (una cucharada para 1 vaso de agua.)
Para alcalinos: Leche, agua, cualquier jugo de fruta o vinagre.
Para pacientes de 1 - 5 años — 1 a 2 vasos.
Para pacientes de 5 años o más — hasta 1 litro.

Induzca al vómito cuando se ingieren materias no corrosivas:

1. Administre leche o agua (para pacientes de 1 - 5 años — 1 a 2 vasos) (Para pacientes de más de 5 años, hasta 1 litro)
2. Provoque el vómito colocando la parte roma de una cuchara o su dedo en la parte de atrás de la garganta del paciente, o puede usar este emético: 2 cucharadas de sal en un vaso de agua tibia. Cuando empiece la náusea y el vómito, coloque al paciente boca abajo con la cabeza más baja que las caderas. Esto evita que el vómito entre a los pulmones y cause daño adicional.

II. Veneno que se inhala:

1. Cargue al paciente (no le permita caminar) y llévelo a sitio de aire fresco inmediatamente.
2. Abra todas las ventanas y puertas.
3. Afloje las ropas apretadas.
4. Aplique respiración artificial si la respiración ha cesado o es irregular.
5. Evite enfriamiento (cubra el paciente con frisas).
6. Mantenga al paciente lo más quieto posible.
7. Si el paciente tiene convulsiones, manténgalo en cama en cuarto semioscuro; evite ruido y movimientos alrededor del paciente.
8. No dé alcohol en forma alguna.

III. Contaminación de la piel:

1. Lave la piel con agua corriente (ducha, manga o pluma de agua)

2. Aplique corriente de agua a la piel mientras quita la ropa.
3. Limpie la piel con agua, la rapidez en el lavar es muy importante para reducir la extensión del daño.

IV. Contaminación a los ojos:

1. Mantenga los párpados abiertos, lave los ojos con una corriente suave de agua inmediatamente, una demora de pocos segundos aumenta grandemente la extensión del daño.
2. Continúe el lavado hasta que llegue el médico.
3. No use substancias químicas, ésta puede aumentar la extensión del daño.

V. Personas inyectadas (picadas de escorpión o de culebras)

1. Haga que el paciente se acueste tan pronto como sea posible.
2. No dé alcohol en forma alguna.
3. Aplique torniquete más arriba del sitio de la picadura (por ejemplo entre brazo o pierna y el corazón). El pulso en los vasos más abajo del torniquete no debe desaparecer, ni debe el torniquete producir una sensación de pulsación. Debe aflojarse el torniquete por 1 minuto a intervalos de 15 minutos.
4. Aplique bolsa de hielo al sitio de la picada.
5. Cargue al paciente para llevarlo al médico o al hospital. NO LE PERMITA ANDAR.

VI. Quemaduras causadas por substancias químicas:

1. Use gran cantidad de agua corriente (excepto en las causadas por fósforos)
2. Cubra el área inmediatamente con un paño limpio que quede flojo.
3. Evite el uso de ungüentos, grasas, polvos, y otras drogas en la primera ayuda del tratamiento de quemaduras.
4. Trate el choque (shock) manteniendo al paciente acostado, manteniéndolo en calor y tranquilo hasta que llegue el médico.

Medidas para la prevención de accidentes por envenenamiento

A. Mantenga todas las drogas, substancias venenosas y substancias químicas para uso en el hogar, fuera del alcance de los niños.

B. No almacene productos **no** alimenticios en las mismas tablillas usadas para guardar los alimentos.
C. Mantenga todas las substancias venenosas en sus recipientes originales; no los cambie a recipientes sin rotular.
D. Cuando tenga que eliminar medicinas, destrúyalas. No las eche en sitios donde puedan ser alcanzadas por niños o por animales domésticos.
E. Cuando dé a los niños medicinas con buen sabor o con olores vivos, siempre refiérase a ellos como medicina, nunca se le debe decir que es dulce.
F. No tome ni dé medicinas en la oscuridad.
G. **LEA LOS ROTULOS ANTES DE USAR CUALQUIER PRODUCTO QUIMICO.**

EDITORIAL

LA SOCIEDAD PUERTORRIQUEÑA DE MEDICINA INTERNA
(The Puerto Rico Society of Internal Medicine)

Es una realidad innegable que la función que ejerce el internista en la Medicina, no es debidamente comprendida en nuestro país. Hubo una vez, cuarenta años atrás más o menos, cuando se solicitaba el consejo de un "clínico" en una consulta. Pero en el día de hoy, ese "clínico" que es continuado y personificado en el internista, no es reconocido por nuestro pueblo. Y cuando digo pueblo, no me refiero solamente a individuos, o a familias, sino también a instituciones responsables de planes de seguro médico, y a gobiernos responsables de la aprobación de leyes que **no** brindan protección adecuada a aquellas personas que se acogen a dichos planes.

¿Y qué es lo que ha pasado? Pues sencillamente que ahora se depende del gastroenterólogo, cardiólogo, y demás sub-especialistas dentro de la especialidad de la Medicina Interna, quedando el internista casi totalmente ignorado. Y así sucede que aquel médico que sacrificó varios más de sus años jóvenes que otros, para poder llegar a ser un Internista, al llegar a nuestro país tiene que anunciarse en, y dedicarse a una sub-especialidad si quiere salir relativamente airoso en nuestra vida profesional. Esto conlleva la pérdida gradual pero casi obligada de conocimientos adquiridos mediante estudio en otros aspectos de la especialidad de Medicina Interna que tanto trabajo le costó obtener durante su entrenanamiento.

Lo mismo, aunque no a igual extremo, ha sucedido en los Estados Unidos. El "American College of Physicians" creó un comité para que se ocupara de buscar una solución a este problema. Pero no pasó mucho tiempo sin que saliera a relucir el hecho de que hacía falta algo más que "un comité" para poder luchar contra el monstruo que se estaba "tragando" al Internista. Había que crear no un simple comité, sino una organización que se dedicara **solamente** a encarar esos problemas que tienden a hacer desaparecer la Medicina Interna como especialidad. Así nació "The American Society of Internal Medicine" (ASIM) el 7 de abril de 1957 cuando 27 sociedades de distintos estados se unieron, reconociendo la necesidad que había de que la nación americana hiciera más y mejor uso de los servicios del Internista para beneficio de la salud de su pueblo.

Algunos de nuestros colegas en Puerto Rico ya han iniciado una campaña similar fundando "The Puerto Rico Society of Internal Medicine". En la reunión anual del ASIM llevada a cabo el

27 de abril de 1958 en Atlantic City, fué aceptada como miembro de la Federación. El pertenecer a esta organización ya ha rendido beneficios en la determinación de honorarios médicos para el Internista en el programa de Medicare y en el Veterans Home Care Program.

Es nuestra intención, a través de estas líneas, hacer un llamamiento a los médicos que están interesados en que nuestro pueblo tenga pleno conocimiento de lo que significa la palabra internista. Esta Sociedad es la organización que cuenta con el mejor respaldo para hacerle frente a los problemas sociales, políticos, y económicos que amenazan el futuro de esta especialidad. Hay mucho por delante que hacer y se necesita el mayor número posible de miembros que estén dispuestos a aceptar el reto.

Es obvio que el médico que desee ser admitido en esta Sociedad ha de llenar varios requisitos, pero el más importante de ellos es el que ejerza **exclusivamente** la especialidad de Medicina Interna. No es nuestra intención hacer uso de estas páginas para dar todos los detalles que el solicitante debiera conocer. Sin embargo, dicha información puede ser obtenida del Secretario de la Sociedad, el doctor Ernesto C. Martínez.

SECCION ADMINISTRATIVA

CARTA MENSUAL DEL PRESIDENTE

Reunión Ordinaria de la Cámara de Delegados: En la tarde del sábado, 12 de abril de 1958, se llevó a efecto en el domicilio de la Asociación Médica la primera reunión ordinaria del año en curso de la Cámara de Delegados, bajo la presidencia del doctor Enrique Pérez Santiago. A esta primera reunión asistieron los siguientes delegados:

Por la Directiva Central:

Luis R. Guzmán López
E. Fernández Cerra
José A. de Jesús
Guillermo Picó

Por el Distrito Este:

Eladio A. Montalvo
Agustín M. de Andino
E. S. Colón Rivera
Héctor Feliciano
Pedro J. Collazo
Víctor M. Rivera
Oscar Costa Mandry
Antonio Rullán
Bernabé Lima Báez
Héctor M. Sampayo
E. Pérez Santiago
Ramón A. Sifre
Jaime F. Pou
Angel M. Mattos
Carlos Guzmán Acosta
F. Hernández Morales
Gualberto Rabell
José Berio
Francisco Berio
Ricardo F. Fernández
J. Basora Defilló
Pablo Luis Morales
José S. Licha
Fernando Vallecillo
R. Cuevas Zamora
José A. Peña
Rafael A. Gil

Por el Distrito Norte:

Z. Rivera Biascoechea
Julio A. Santos

Por el Distrito Sur:

Carlos Jiménez Torres
Luis A. Maduro
José Luis Jiménez

Por el Distrito Oeste:

J. Ramírez Ledesma
José Echegaray
Paul E. Kindy

Presentaron sus excusas por no poder asistir a la reunión los siguientes delegados: C. José Ferraioli, Armando Antommattei, A. Otero López y A. García Soltero.

La Cámara conideró los siguientes asuntos:

1. Fué leída y aprobada el acta correspondiente a la reunión ordinaria celebrada en noviembre de 1957.

2. **Comité de Resoluciones**: El señor presidente de la Cámara nombró el siguiente Comité de Resoluciones:

   J. Basora Defilló, Pres.
   Jaime F. Pou
   Paul E. Kindy
   Gualberto Rabell
   C. Jiménez Torres
   Julio A. Santos
   José Berio

3. **Comité de Finanzas**: Fueron nombrados asimismo los siguientes compañeros para constituir el Comité de Finanzas de la Cámara:

   José S. Licha, Pres.
   Fernando Vallecillo
   Z. Rivera Biascoechea
   Antonio Rullán
   Luis F. Sala
   José A. Peña
   José Echegaray

4. Acto seguido esta presidencia presentó un amplio informe en cuanto a la legislación de carácter médico que está ante la consideración de la Asamblea Legislativa.

5. A continuación se dió cuenta de las proposiciones para un seguro de vida grupal hechas por las firmas Pan American Life Insurance y Puerto Rican Life Insurance Company.
   En relación con este asunto la Cámara adoptó el siguiente acuerdo:

   (a) Expresarse a favor de que se extienda la cuantía del seguro que cubre actualmente a los médicos asociados.
   (b) Referir las proposiciones recibidas a la Junta del Auxilio Médico Mutuo para su estudio y recomendaciones pertinentes en la próxima reunión.
   (c) Autorizar al presidente para que haga una encuesta entre la matrícula para determinar la disposición de los miembros a coger este tipo de seguro.

6. Carta del doctor J. Ramírez Ledesma en relación con los anuncios de medicinas de elaboración dudosa que contínuamente se hacen por la radio, la Televisión y la Prensa del país.
   En relación con este asunto se acordó consultar al Asesor Legal de la Asociación para determinar ante qué agencia estatal o federal deberá plantearse este problema.

7. Planteamiento de la Directiva en relación con el propuesto

cargo de Director Ejecutivo, para cuya creación se recomienda un aumento de $20.00 en la cuota.
Este asunto quedó sobre la mesa para ser discutido ampliamente en una reunión extraordinaria de la Cámara que se celebrará próximamente.

8. Enmienda al Reglamento: Se aprobó una enmienda a la Sección Tercera del Capítulo VIII del Reglamento, para que en lo sucesivo el Comité de Credenciales sea conocido como "Comité de Credenciales y Admisión de Socios", de manera que además de verificar las credenciales del candidato a socios, el Comité quede facultado para investigar las condiciones morales y las relaciones del candidato.

9. Se acordó respaldar la actitud de la Asociación Médica del Distrito Sur de enviar una comunicación a los miembros del distrito llamándoles la atención hacia el hecho de que asociarse con una compañía de optómetras es antiético y el médico que así actúe está sujeto a ser expulsado de la Asociación.

❖ ✦ ❖

Asamblea Anual Asociación Médica del Dtto. Este: La Asociación Médica del Distrito Este, que preside el doctor Antonio Rullán celebrará su asamblea anual el viernes 23 y sábado 24 de mayo, en el domicilio de nuestra Asociación.

Bajo la presidencia del doctor Egidio S. Colón Rivera, el Comité Científico del distrito ha estructurado el siguiente programa:

Viernes, 23 de mayo

8:30 p.m. Management of some Congenital Deformities, W. J. Benavent, M.D.
9:00 Primary Tuberculosis in Children, José E. Sifontes, M.D.
9:30 Evaluation of the Prenatal Period, Rafael A. Gil, M.D.

Sábado, 24 de mayo

11:00 a.m. Proyección de películas
(a) The Doctor Defendant (b) The Medical Witness
2:30 p.m. Symposium: Problemas de interés médico, legal y económico.
(a) Etica Médica, Luis A. Sanjurjo, M.D.
(b) Planes de Seguro médico prepagado, Jaime F. Pou, M.D.
(c) Contribuciones sobre Ingresos, Sr. Ramón H. Vélez.

(d) Responsabilidad Profesional, Lcdo. F. Ponsa Feliú.

El Comité de Convención, presidido por el doctor Miguel Valiente, ha organizado los siguientes actos de carácter social:

Viernes: 10:00 p.m.—Agape en el Club Médico
Sábado: 12:30 p.m.—Buffet en la Asociación Médica
8:00 p.m.—Banquete-baile en el Caparra Country Club

El programa de la asamblea circulará próximamente entre la matrícula. Le recomendamos que desde ahora haga arreglos para asistir a estos actos.



Pago de Cuota: El señor Tesorero, doctor C. José Ferraioli, nos pide recordemos a la matrícula que el plazo para el pago de la cuota venció el 31 de marzo pasado. Se suplica a aquellos compañeros que aún no han satisfecho su cuota procedan a hacerlo a la mayor brevedad posible. Nuestra Asociación necesita la entusiasta cooperación de la matrícula para poder seguir adelante con su labor.



Sensible Fallecimiento: El doctor Luis A. Sanjurjo, ha tenido la gentileza de comunicarnos el fallecimiento del distinguido urólogo americano, doctor Thomas D. Moore, miembro honorario de la Asociación Puertorriqueña de Urología, y quien en varias ocasiones visitó nuestra Isla y dictó muy interesantes conferencias ante la matrícula de nuestra Asociación.

Vaya nuestra expresión de condolencia para los atribulados deudos de este buen compañero.

❖ ✦ ❖

Programa Medicare: Ya ha sido distribuído entre los compañeros que han prestado servicios al Programa Medicare el nuevo Manual y Lista de Honorarios que rige desde el 1ro. de febrero del año en curso.

Si usted no ha recibido aún su copia o si está interesado en rendir servicios bajo este programa en el futuro solicite el nuevo SCHEDULE OF ALLOWANCES del Comité de Medicare, que preside el doctor Jaime F. Pou.

❖ ✦ ❖

Liga Puertorriqueña Contra el Cáncer: La Liga Puertorriqueña Contra el Cáncer, cuya magnífica labor es de todos conocida, pero muy especialmente de la clase médica puertorriqueña, está llevando a cabo su campaña anual de recolección de fondos.

Una vez más exhortamos a nuestra matrícula a contribuir, a la medida de sus recursos, a esta noble institución, genuinamente puertorriqueña, que debe su existencia al espíritu de sacrificio, el

entusiasmo y la generosidad de nuestro querido expresidente fenecido, doctor Isaac González Martínez, q.e.p.d.

❖ ✦ ❖

Proyectos del Escudo Azul: A la fecha de circulación de esta Carta Mensual los proyectos de ley que autorizan la organización del programa de Escudo Azul, que con tanto tesón ha defendido nuestra Asociación, han sido ya aprobados en votación final por la honorable Cámara de Representantes de Puerto Rico.

Hay magníficos indicios de que próximamente el honorable Senado pase a considerar dichas medidas legislativas, y confiamos que también les imparta su aprobación.

Se acerca el final de la primera etapa y si ésta se ve coronada por el éxito, se abrirá una segunda jornada, todavía más árdua, en que todos los compañeros, hombro con hombro, trabajaremos en la estructuración de este programa de servicios médico-quirúrgicos para la clase media, que tanto significa para nuestro pueblo.

❖ ✦ ❖

Seminario de Medicina Industrial: Hemos visto, con verdadero agrado, el interesante programa que ha preparado la Comisión Industrial de Puerto Rico, a través de su Director Médico, el buen compañero Dr. Hirám Vázquez Milán, y el cual se llevará a efecto en esta ciudad durante los días 26, 27, 28 y 29 de mayo.

El programa preliminar ya está circulando entre los compañeros asociados. Los tópicos seleccionados para este seminario son de sumo interés, y nos complacemos en exhortar a todos para que asistan al mayor número de actos que les sea posible.

❖ ✦ ❖

Aviso de Gran Interés: Nos complacemos en transcribir a continuación parte de una comunicación que nos ha enviado el doctor J. Serra-Chavarry, Director de la Administración de Veteranos en Puerto Rico:

> "As you probably know, we recently received full approval for a three-year residency training program in Physical Medicine & Rehabilitation.
>
> "Since physiatrists are scarce personnel, the Veterans Administration has converted all fully approved PM&R residency training programs into Career Residencies. This means that physicians selected for this type of training receive the salary that any physician in regular employment would receive as recommended by the Professional Standards Board. Naturally, the Veterans Administration requires that these phy-

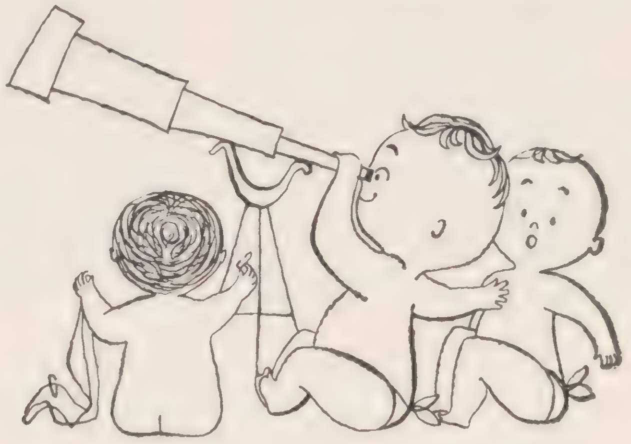
sicians serve the VA for a number of years after they finish their training."

Los compañeros interesados pueden comunicarse con el doctor Serra-Chavarry.

Luis R. Guzmán-López, M.D.
Presidente



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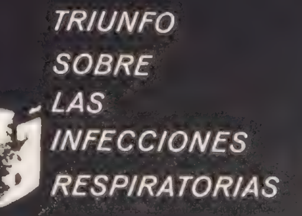
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HYPERTHYROIDISM AND PREGNANCY:

A CLINICAL STUDY ON THE EFFECTS OF ANTITHYROID DRUGS*

*AGUSTIN M. DE ANDINO JR., M.D.***

Severe hyperthyroidism usually gives rise to sterility in the affected female and because of that, it is a rather rare complication of pregnancy. The reported incidence of hyperthyroidism in pregnancy varies between 0.008 to 1.4%, with an average figure of about 0.2%.[1-11] For many years this complication had been viewed with considerable alarm because of the high incidence of miscarriage and stillbirth, but as treatment for hyperthyroidism has improved, there has been less basis for fear. This condition posed a difficult therapeutic problem both for the internist and the obstetrician, for which, until lately the only solution available was the surgeon's knife under unfavorable conditions or irradiation of the thyroid gland. In 1929 Gardiner-Hill[12] reported that 50% of the pregnancies complicated by hyperthyroidism ended unsuccessfully. Other reports during the last thirty years[5,13,14,15] on the treatment of hyperthyroidism during pregnancy with Lugol's solution, with or without subtotal thyroidectomy, have indicated that the incidence of miscarriage or stillbirth is reduced by therapy but still remains higher in thyrotoxic patients. With the advent of potent antithyroid drugs the outlook of this condition has changed considerably and the internist must now share with the obstetrician the care of the pregnant patient with thyrotoxicosis.

CLINICAL MATERIAL AND METHODS

In this paper are presented our experiences in the medical management of eight pregnant thyrotoxic patients followed at the Endocrine Clinics of the San Juan City Hospital and the Presbyterian Hospital between July 1, 1951 and July 1, 1957. These eight

---

* Presented before the Regional Meeting of the American College of Physicians, San Juan, P. R. October 16, 1957.

** From the Department of Medicine and the Endocrine Clinic, San Juan City Hospital, and the Department of Medicine, School of Medicine, University of Puerto Rico, San Juan, Puerto Rico.

TABLE I: INCIDENCE OF HYPERTHYROIDISM IN PREGNANCY

Author	Year	Region	No. of Preg-nancies	No. of Cases Complicating Pregnancy	% Incidence
Markoe[1]	1918	New York	100,000	8	0.008
Wallace[2]	1933	Brooklyn	11,571	9	0.077
Bustos Morón[3]	1935	Buenos Aires	30,000	5	0.016
Portis and Roth[4]	1939	Chicago	1,000	14	1.4
Javert[5]	1940	New York	23,439	18	0.076
Baumgartner[6]	1942	Los Angeles	1,585	22	1.39
Mc Laughlin and Mc Googan[7]	1942	Omaha	6,112	19	0.31
Davis[8]	1944	Baltimore	20,000	8	0.04
Kibel[9]	1944	New York	15,864	12	0.075
Ciulla[10]	1949	Pavia, Italy	10,202	18	0.176
Dailey and Benson[11]	1951	San Francisco	11,390	21	0.175
de Andino	1951-1957	San Juan, P. R.	25,377	10	0.039

patients represent a total of ten pregnancies. The patients were seen by us at different stages of their pregnancy. Three became pregnant while under strict observation at the Clinic while the others came to us at different months of gestation with the initial onset or a recurrence of their thyroid disease. Their age varied between 22 and 37 years; four were white and four were colored. One of them (T. de J.) also exhibited thyrotoxic heart disease with congestive failure and multiple uterine fibroids as a complication of her first pregnancy. On her two other pregnancies the heart disease was well under control. All patients were treated routinely with antithyroid medication. Methylmercaptoimidazole (Tapazole) was used in all excepting case No. 1. (T. de J.), who received propylthiouracil during her second pregnancy. The starting dose was usually 10 mgm. every six hours; this dose was rapidly reduced as euthyroidism was attained. The dose of Tapazole was kept at a level so as to insure a BMR of +20 to +25% during the last trimester of pregnancy. Patients were observed at biweekly intervals during the second and third trimester of pregnancy and special attention was given to the prevention of hypothyroidism. In the last four patients studied Lugol's solution was added to the therapeutic regime during the last trimester of pregnancy.

CLINCAL OBSERVATIONS

The pertinent clinical findings appear in Table II. In case No. 1 (T. de J.) the amount of Tapazole used in the control of

her thyrotoxic condition during her first pregnancy was insufficient. The initial dose was 5 mgm. three times daily, and was gradually reduced to 10 mgm. daily. In the light of our present knowledge we now know that this dose is insufficient to control any degree of thyrotoxicosis.[22] The patient also exhibited evidence of severe thyrotoxic heart disease with congestive failure, and in addition had multiple fibromyomata uteri. The combination of these effects culminated in a stillbirth at 6 months. During her second pregnancy her thyrotoxicosis was well controlled with propylthiouracil using 600 mgm. as a starting dose. This dose was gradually reduced to 200 mgm. daily in divided doses up to the day of delivery. This pregnancy yielded a premature male child in good state of health. A third uneventful pregnancy was achieved when she was in a state of remission following an adequate course of propylthiouracil.

The second case (S. M.) became pregnant after her disease had been controlled under Tapazole therapy. After being followed in the Clinic for a period of about a year she disappeared from sight but continued taking Tapazole on her own accord in a dose of 5 mgm. three times daily. She was seen by us in her 6th month of pregnancy and the antithyroid medication was continued in doses of 20 mgm. daily. Her BMR in the last trimester of pregnancy ranged between +15 and +25%. She had an uneventful delivery giving birth to full term identical female twins. The children did not exhibit any abnormality.

The third case (B.P.M.) was a case of recurrent thyrotoxicosis which was seen by us for the first time in her fourth month of pregnancy. She had been in a state of remission after subtotal thyroidectomy which had been performed two years prior to admission. Her thyrotoxicosis was mild and she was well controlled with 20 mgm. of Tapazole daily. On November 1952 she delivered a full term male infant who exhibited no abnormalities.

The fourth case (M.C.I.) was first seen by us in her sixth month of pregnancy. At that time she gave a history of thyrotoxicosis which could be traced to about sixteen months prior to admission. After the diagnosis was confirmed she was immediately started on 30 mgm. of Tapazole daily and one drop of Lugol's solution three times daily. While under observation at the Endocrine Clinic the dose of Tapazole was rapidly reduced to 15 mgm. daily. She gave birth to a normal full term female child without any complications.

The fifth case (N.R.S.) was a 24 year old female which had been under treatment with antithyroid medication at the Endocrine Clinic of the Presbyterian Hospital since November 7, 1950. On her visit on March 28, 1953 it was found that she was in her

first month of pregnancy. At that time her toxic symptoms were well under control with 20 mgm. of Tapazole daily. The dose of Tapazole was rapidly reduced to 15 mgm. daily and during her last month of pregnancy she only received 10 mgm. daily. On November 14, 1953 she delivered a normal full term female infant without any difficulties. The physical examination of the child was within the limits of normal.

The sixth case (G.F.V.) in this series was that of a 26 year old white female which had been under therapy for thyrotoxicosis since June 1, 1951. She had been under antithyroid medication off and on for a period of three years. Prior to conception she had been under excellent control with 20 mgm. of Tapazole daily. On July 5, 1954 she reported to us with the chief complaint of amenorrhea of two months duration. The diagnosis of pregnancy was confirmed by the Obstetrical Department. Lugol's solution in a dose of three drops three times daily was then added to the therapeutic regime. The dose of Tapazole was gradually reduced to 10 mgm. daily. On February 8, 1955 she gave birth to a normal full term male infant. The physical examination of the child did not reveal any abnormalities.

The seventh case (B.S.R.) was extremely interesting. This 27 year old colored female, gravida VI and para III, was admitted at term to the Obstetrical Department of the San Juan City Hospital on March 9, 1956 with a history of premature rupture of the membranes of 36 hours duration. The patient gave a history suggestive of hyperthyroidism dating back to one year prior to admission. She had been under inadequate antithyroid therapy off and on for this period of time. The patient had discontinued all therapy on her own during the month prior to admission. The physical examination revealed the classical findings of hyperthyroidism together with a blood pressure of 180 90 and +2 pretibial edema of both legs. She was immediately placed on a regime consisting of 50 mgm. of Tapazole daily in divided doses together with 5 drops of Lugol's solution three times daily in combination with a low salt diet. On March 13, 1956 four days after admission, the patient was delivered of a normal female child with no demonstrable physical abnormality. The post partum period was uneventful.

The eighth case (C.R.P.) was seen in our Endocrine Clinic in the fifth month of pregnancy with the chief complaint of swelling of the anterior part of the neck, difficulty in swallowing, nervousness, palpitations and insomnia of three months duration. The physical examination revealed the classical findings of a diffuse exophthalmic goiter. The laboratory studies confirmed the clinical diagnosis. The patient was then started on 30 mgm. of Tapazole

TABLE II: EFECTS OF ANTITHYROID MEDICATION IN PREGNANCY COMPLICATED BY HYPERTHYROIDISM

No.	Patient	Gestation (Mo.)	Drug	Child Condition	Goiter	Thyroid Status During Pregnancy	Complications
1. (a)	T. de J.	5th mo.	Tapazole	Stillbirth male	No	Severely toxic first 5 mos. Not controlled. Insufficient dose.	Thyrotoxic heart disease with failure. Fibromyomata uteri.
(b)	" "	3rd mo.	Tapazole (5 mos.) Propyl. (3 mos.)	Premature male	No	Euthyroid (5th mo.)	Fibromyomata uteri
(c)	" "	4th mo.	None	Normal male	No	Euthyroid (after treatment)	Fibromyomata uteri
2.	S.M.	6th mo.	Tapazole	Identical female twins	No	Mildly toxic first 6 mos.	None
3.	B P.M.	6th mo.	Tapazole	Normal male	No	Recurrent thyrotoxicosis (4th mo.)	None
4.	M.C.I.	6th mo.	Tapazole	Normal male	No	Mildly toxic (6th mo.)	None
5.	N.R.S.	1st mo.	Tapazole	Normal male	No	Euthyroid thru pregnancy	None
6.	G.F.V.	2nd mo.	Tapazole	Normal male	No	Euthyroid thru pregnancy	None
7.	B R.S.	Term	Tapazole	Normal male	No	Toxic when delivered	Pre-eclampsia
8.	C.R P.	[illegible] mo.	Tapazole	Premature male	No	Mildly toxic (6th mo.)	Abruptio placenta and prolapse of umbilical cord (7th mo.)

	FETAL MORTALITY	9.09%
CORRECTED	FETAL MORTALITY	0%
	MATERNAL MORTALITY	0%

given in divided doses during the day and night supplemented by an adequate amount of vitamins of the B-complex group. On the early part of the last trimester of pregnancy Lugol's solution in a dose of 5 drops 3 times daily was then started. On the 7th. month of pregnancy she was admitted as an emergency case to the Obstetrics Department with the diagnosis of abruptio placenta and prolapse of the umbilical cord. An emergency Cesarean section was performed and the patient was delivered of a 3½ lbs. premature male infant. The physical examination of the child was within the limits of normal. The mother tolerated the surgical procedure quite well and had an uneventful convalescence. Up to date both mother and child are doing fine.

DISCUSSION

In the older medical literature one finds the suggestion more than once that both simple and toxic goiter make their initial appearance during the pregnant state. Although this might be the case with simple goiter the studies of Mussey and associates[14] and Astwood[16] indicate that this rarely occurs with hyperthyroidism. This last view is well supported by our series since in only one of our cases did it appear as if the hyperthyroidism had its onset during pregnancy.

From a perusal of the modern literature it appears as if the older view that hyperthyroidism is a grave complication of pregnancy is no longer true. Mussey,[17] Clute and Daniels,[18] and Dailey and Benson[11] hold that hyperthyroidism per se produces no adverse effect upon a well established pregnancy. These observers claim that the occurrence of complications during pregnancy is not much higher in hyperthyroidism or in hyperthyroidism treated with subtotal thyroidectomy than in normal pregnancy. In the series of cases reported by Astwood[16] he concluded that pregnancy exerted a beneficial effect upon the hyperthyroid state. As a matter of fact, not only was the disease controlled by the usual dose of the antithyroid drug or by a lower dose, but the incidence of lasting remissions was quite high after the pregnancy ended. Case No. 7 in our series illustrates their point quite well as the thyrotoxic state did not interfere with the completion of the pregnancy with the delivery of a normal child.

What is the effect of the thyrotoxic state upon the developing infant? In our review of the literature on this subject we have not been able to find any particular fetal abnormality known to be caused by maternal hyperthyroidism and no placental aberrations definitely attributable to the hyperactive maternal thyroid. Clute and Daniels[18] are of the opinion that hyperthyroidism does

not affect the fetus adversely. On the other hand the fetal loss associated with untreated hyperthyroidism has been found to be quite high. Gardiner-Hill[12] found that 54.5% of such pregnancies terminated unsuccessfully. The fetal loss associated with hyperthyroidism treated by subtotal thyroidectomy has also been found to be quite high varying between 24 and 33%.[11,19]

Most important is the possible adverse effects of thiouracil and its derivatives upon the fetus. In a recent review of the literature Elphinstone[20] found only eleven reported cases of abnormalities in the fetus or in the newborn which could be directly abscribed to the antithyroid medication. He contributed a case of his own. This is quite encouraging in view of the fact that experimental use of thiouracil in pregnant rats has resulted in activation and hyperplasia of the thyroid gland with retarded growth of the offspring. These changes are temporary if treatment with the drug is stopped.

Freisleben and Kjerulf-Jensen[21] in experiments on rats proved that thiouracil compounds are freely transmitted from mother to fetus through both the placenta and the breast milk. Furthermore, the hyperplasia of the thyroid in infant rats can be prevented by the current administration to the mother of thyroid hormone along with the thiouracil derivative. They concluded that the danger is not from the thiouracil derivative but from the low content of thyroxin in the tissues.

Our observations coupled with other reports in the medical literature and specially that by Astwood[16] in 1951 suggest that antithyroid therapy can be safely continued thru pregnancy without harm to either mother or child provided certain precautions are taken, specially in regards to prevention of hypothyroidism. Astwood reported a series of 22 completed pregnancies yielding 22 living children who showed no evidence of thyroid disturbance or goiter. Three of these children were premature. His fetal mortality was 0%. Our series show a corrected fetal mortality of 0% and two premature births. These results compare favorably with those obtained by Dailey and Benson[11] and by Bell[19] in their respective series of cases treated by subtotal thyroidectomy during pregnancy. Their fetal mortality was 24% and 33% respectively.

SUMMARY

1) We have reported our experiences in a series of eight pregnant thyrotoxic patients treated with antithyroid medication. The ten completed pregnancies yielded a total of eight normal infants, two premature ones and one stillbirth.

2) The corrected fetal mortality was 0%. The maternal mortality was 0%.

3) When antithyroid drugs are used during pregnancy it is imperative to use a full therapeutic dose in order to restore metabolic equilibrium as soon as possible. After this state is achieved the dose should be rapidly reduced in order to avoid hypothyroidism, as this latter state increases the risk of abortion in early pregnancy and in late pregnancy might give rise to congenital cretinism and goiter formation. The addition of iodine in the last trimester of pregnancy is of help in the prevention of goiter formation in the child.

4) If the antithyroid medication is continued after delivery it is imperative that lactation of the infant by the mother be avoided as the antithyroid compounds diffuse freely into the mother's milk.

5) Our observations suggest that antithyroid therapy can safely be used throughout pregnancy without harm to mother or child.

6) We agree with Astwood's observation on the fact that pregnancy appears to exert a beneficial effect on the hyperthyroid state.

ACKNOWLEDGEMENTS

We wish to thank Dr. Manuel Paniagua for his kindness in allowing us to follow two of his cases at the Endocrine Clinic of the Presbyterian Hospital. We also thank Dr. Roberto Busó for his wholehearted cooperation in performing 24 hr. radioiodine studies in some of these patients at the Fundación de Investigación Clínicas, Hospital Mimiya, Santurce.

ADDENDUM

After this paper was prepared one month ago we have had the opportunity of seeing two more patients in which the problem of hyperthyroidism complicating pregnancy had arisen. One case, a 29 year old white female patient with a diagnosis of Graves' disease and pregnancy was seen at the Medical Clinic of the Presbyterian Hospital through the courtesy of Dr. Manuel Paniagua. She was safely carried thru pregnancy with a maintainance dose of 20 mgm. of Tapazole daily and had an uneventful delivery without complications. The child was a perfectly normal one and did not exhibit deformities or goiter. The second patient happens to be quite an exciting one. This the case of a 20 year old white female who forms part of a group of thyrotoxic patients in which

the action of Reserpine as a therapeutic agent has been under investigation. She was initially seen at our Endocrine Clinic on October 25, 1956 with the chief complaint of bulging of the eyes, weight loss, palpitations, headaches and dizziness of 3 months duration. The physical examination revealed the classical findings of a diffuse exophthalmic goiter and the laboratory studies confirmed the diagnosis. She was initially started on Tapazole but in a period of three weeks she developed a rash over the abdomen and the back. On December 20, 1956 Tapazole was discontinued and Reserpine was started in a dose of 0.1 mgm. four times daily. On her next visit on March 14, 1957 there was remarkable improvement in her clinical condition with total disappearance of all subjective complaints. She disappeared from the Clnic and did not return until September 5, 1957 at which time she gave us the history of being in her six month of pregnancy. She had continued therapy with Reserpine on her own until the early part of July, 1957. It was obvious that she had become pregnant towards the latter part of March, 1957 at which time she had gone into a remission of her disease as a result of Reserpine therapy. Clinical and laboratory evaluation revealed that she was in a state of euthyroidism. This patient represents the first instance recorded in medical literature of a female patient with hyperthyroidism who became pregnant when her disease was brought into control through the action of Reserpine.

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HEMOPHILIA AND HEMOPHILIA-LIKE SYNDROMES

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Hemophilia presents a well known syndrome. With the advent of new diagnostic laboratory procedures the classification of these patients into various groups has been made possible.[1]

Utilizing these newer techniques a group of patients have been restudied aiming to elicidate the type of coagulation defect. These patients originally came to the San Juan City Hospital with the chief complaint of bleeding of the deep intramuscular type. The clinical picture in all of them suggested a diagnosis of hemophilia. The present communication describes the result of this work.

Materials and Methods: Seventeen patients hospitalized in the San Juan City Hospital for a hemorrhagic diathesis have been studied. The laboratory methods employed were as follows:

Clotting Time — Lee and White

Bleeding Time — Ivy

Plasma Prothrombin — one stage of Quick

Platelets — direct method

Prothrombin Consumption[2]

Circulating anticoagulants[3]

Mixtures of the patients plasma with the plasma and serum of known hemophilics, as well as with normal plasma, absorbed with barium sulfate and normal serum were utilized to classify the patients into various groups. (Table I)

Discussion and Results: The first step in blood coagulation depends upon the formation of available plasma thromboplastin from inactive plasma precursors (A.H.G., P.T.C., P.T.A. and others) and the interaction of Platelet Thromboplastic factor. (Figure 1).

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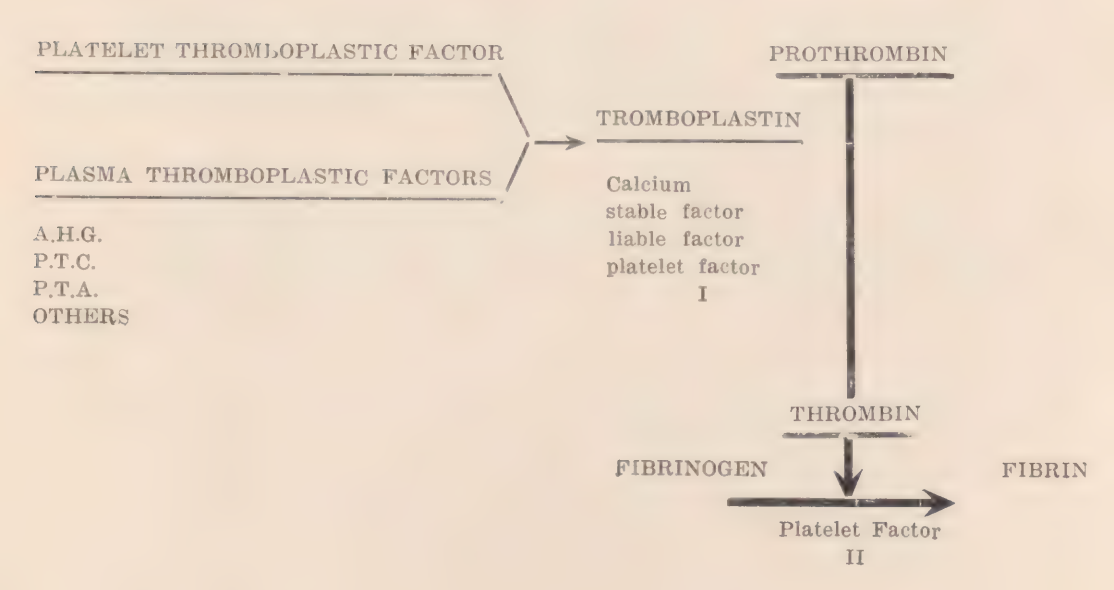
Table I

IDENTIFICATION OF TYPE OF HEMOPHILIA

A. Coagulation defect corrected by:	Normal Plasma	$BaSO_4$ Plasma	Normal Serum
Hemophilia A Classic	+	+	—
Hemophilia B Christmas	+	—	+
Hemophilia C PTA	+	+	+
Circulating Anticoagulants	—	—	—
B. Mixtures with bloods of known Hemophiliacs			

Figure 1

SCHEMATIC REPRESENTATION OF BLOOD COAGULATION



A deficiency of available thromboplastin may be due to:[4,5,6,7,8,9,10,11]

1. Insufficient formation of plasma thromboplastic precursors.
   a. Hereditary deficiencies

Factor	Disease
A.H.G.	Hemophilia A - Classic
P.T.C.	Hemophilia B - Christmas Disease
P.T.A.	Hemophilia C

others: P.T.F. - D
Hageman
Factor X
Stuart

   b. Acquired deficiencies

II. Inactivation or destruction of thromboplastin or thromboplastic precursors by circulating anticoagulants.

Deficiency of thromboplastin results in hemorrhagic diathesis characterized by deep intramuscular type of bleeding as seen in classical hemophilia.[12]

Table II illustrates the history and pertinent clinical findings in our group of patients. Patients 1 to 13 had a family history of bleeding. The first hemorrhagic episode was observed at various periods of time dating from birth to 48 months. Although no family history of bleeding was obtained in case 14, the fact that symptoms appeared so early in his life suggests the presence of a congenital defect.

Cases 15, 16, and 17 represent acquired forms. The three were women and their ages were 50, 36, and 43 years, respectively.

The type of bleeding was similar in all patients and characteristic of that seen in hemophilia-like disorders. Epistaxis, hematomas, hematuria, hemarthrosis, G.I. tract bleeding were commonly observed. Petechial bleeding occurred in only one patient as a transitory finding after several blood transfusions.

Basic laboratory findings (Table III) revealed: normal platelet counts, bleeding time and clot retraction, thus eliminating the platelets as the cause of the hemorrhagic diathesis. Normal prothrombin time (very slight prolongations in two patients) excluded a deficiency of prothrombin, stable and labile factor and a marked diminution of fibrinogen levels.

The abnormal laboratory findings of prolonged coagulation,

Table II — CLINICAL FINDINGS

	Age Sex	Age at onset of Bleeding	Heredity or Family History	Epistaxis	Spontaneous Hematomas	Hematomas after Trauma	Hematuria	Hemarthrosis	G. I. Tract Bleeding	Gingival Bleeding after eruption of teeth
1. VOV	12 M	9 mos.	+	+	+	+	—	—	—	+
2. JOV	15 M	1 mo.	+	—	+	+	—	+	—	+
3. JAR	9 M	48 mos.	+	—	+	+	—	—	—	—
4. RJR	13 M	18 mos.	+	+	—	+	+	—	—	—
5. VA	14 M	6 mos.	+	+	+	+	+	+	—	—
6. JV	24 M	6 mos.	+	+	+	+	+	+	—	—
7. JMS	15 M	1 mo.	+	—	+	+	+	+	—	—
8. RMS	8 M	at Birth	+	—	+	+	—	+	—	—
9. OR	13 M	18 mos.	+	+	+	+	+	—	—	+
10. JLRV	11 M	6 mos.	+	+	+	+	—	+	—	+
11. DRS	5 M	5 mos.	+	—	+	+	—	—	—	—
12. GA +	8 M	24 mos.	+	+	—	+	—	+	—	—
13. MAGI	2 M	3-4 mos.	+	—	+	—	—	—	—	—
14. WM	14 M	6 mos.	—	+	+	+	—	+	—	+
15. GF	53 F	50	—	—	+	+	+	+	+	+
16. MH +	40 F	36	—	—	+	+	+	+	+	+
17. RR	47 F	43	—	—	+	+	+	—	—	+

plasma recalcification times and abnormal prothrombin utilization pointed towards a diagnosis of hemophilia or hemophilia-like disease. Case 3 with a coagulation time of 7 minutes illustrates the well known fact that a normal clotting time is not incompatible with a diagnosis of hemophilia[12].

Classification of patients into the various types of hemophilia is possible utilizing normal plasma, barium sulfate absorbed plasma and normal serum.

Table III
LABORATORY FINDINGS

	NORMAL				ABNORMAL		
	Bleeding Time (Min.)	Clotting Retraction (Hours)	Platelets	Plasma Prothrombin Control 12-15"	Plasma Recalcification Time (Min.)	Clotting Time (Min.)	Prothrombin Consumption (Sec.)
1. VOV	1-1/4	1	212,000	16	15	50	15
2. JOV	1	1	220,000	14	7-1/2	41	16
3. JAR	1-1/3	1	300,000	15	2-1/6	7	18
4. RJR	1-1/2	1	274,000	15	3	14	16
5. VA	1-1/4	1	264,000	18	9-1/6	51	14
6. JV	2-1/2	1	200,000	17	11	41	17
7. JMS	1-3/4	1	380,000	17	15-1/6	45	15
8. RMS	2-1/2	1	186,000	16	15	46	15
9. OR	10	1	341,000	15	7	12	17
10. JLRV	3-1/2	1	100,000	15	5-5/6	35	12
11. DRS	5	18	88,000	15	5-1/2	20	12
12. GA +	2-2/3	1	156,000	14		28	15
13. MAGI	2	1	164,000	12		18	10
14. WM	2	1	194,000	15	9-1/2	19	15
15. GF	1-1/6	1	152,000	14	10	37	13
16. MH +	2	1	180,000	14	11-1/2	62	11
17. RR	3	1	160,000	16	15	87	12

The results obtained after adding the above mentioned reagents to the blood of the patients under study are seen in Table IV. In five patients the tests were not performed. In seven, the coagulation defect was corrected by normal and $BASO_4$ absorbed plasma and not by normal serum. These were classified as having

Table IV

IDENTIFICATION OF TYPE OF HEMOPHILIA

	DEFECT CORRECTED BY:						
	Normal Plasma	$BaSO_4$ Plasma	Normal Serum	Hemophilia A Plasma	Hemophilia B Plasma	Circulating Anticoagulants	Type of Hemophilia
1. VOV	+	+	−	−	+	−	A
2. JOV	+	+	−	−	+	−	A
3. JAR							
4. RJR							
5. VA	+	+	−	−	+	−	A
6. JV							
7. JMS	+	+	−	−	+	−	A
8. RMS	+	+	−	−	+	−	A
9. OR							
10. JLRV	+	+	−	−	+	−	A
11. DRS	+	±	−	−	−	−	Double def. AHG PTC?
12. GA +							Died
13. MAGI	+	+	−	−	+	−	A
14. WM	+	−	+	+	−	−	B
15. GF	+	−	+	+	−	−	B Acquired
16. MH +	−	−	−	−	−	+	Circulating Anticoagulant
17. RR.	−	−	−	−	−	+	Circulating Anticoagulant

the classical type of hemophilia (hemophilia A). In one patient the laboratory findings were suggestive of a double deficiency of A.H.G. and P.T.C. A similar case has been described by Hill and Speer.[13] Further studies on this case 11 are desirable. Mixtures with the bloods of known hemophilia A and B patients corroborated the above findings.

A deficiency of P.T.C. (Christmas disease) was identified in cases 14 and 15. The first was a young boy with a congenital form of the disease, the second, a 50 years old female patient with an acquired P.T.C. deficiency. This interesting case will be the subject of a more detailed report. Review of the literature fails to reveal a similar case, although acquired diminution of P.T.C. plasma levels are often found associated with alterations in prothrombin concentration during coumarin therapy[14] and in the newborn[15]. Contrary to our case, these patients seem to respond well to vitamin K.

Tests for circulating anticoagulants were negative in the hemophiliacs in whom it was performed. This is surprising in view of the reported frequency of circulating anticoagulants found in such patients as a result of immunization to blood and blood products containing A.H.G. or P.T.C.

The clotting time and prothrombin consumption of patients 16 and 17 were not affected by the addition of normal plasma or serum nor by barium sulfate absorbed plasma. Furthermore, tests for circulating anticoagulants were positive. These two cases have been previously published.[16,3] They represent examples of antithromboplastic anticoagulants. In one the symptoms developed after pregnancy, in the other, they started "de novo" after a tooth extraction. Classification of hemophiliacs into the various types is important from the point of view of treatment as there is a difference in the response to blood and blood products (Table V.)

Table V
RESULTS OF TREATMENT

	Fresh Blood or Plasma	Stored Blood or Plasma	Serum	Survival of coagulation factor
Hemophilia A	+	—	—	12-24 hrs.
Hemophilia B	+	+	+	7 days or more
Hemophilia C	+	+	+	3-7 days
Circulating Anticoagulants	—	— Placental Plasma?	—	

Follow-up of this group of patients reveals that two of the hemophilic patients and patient 16 (circulating anticoagulant) died during episodes of bleeding after trauma. The patient with acquired hemophilia B and patient 17 with circulating anticoagulant are at present normal from the clinical and hematological point of view (3 and 9 years after onset respectively). A very favorable prognosis in acquired forms of the disease is therefore possible.

SUMMARY AND CONCLUSIONS

1. Seventeen patients presenting a hemorrhagic diathesis suggestive of a hemophilia-like disease have been studied.

2. Five patients were not classified as to the type of hemophilia; seven presented the laboratory pattern of classical hemophilia; one was suggestive of a double deficiency of PTC and AHG.

3. Two examples of hemophilia B (Christmas Disease) were found, one congenital and other acquired.

4. Circulating (anti-thromboplastic) anticoagulants were the cause of the hemorrhagic diathesis in two female patients.

5. Two patients with acquired hemophilia-like disease (hemophilia B and circulating anticoagulant) have recovered, and at present, have normal clinical and laboratory findings.

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TRATAMIENTO QUIRURGICO DE VARICES SANGRANTES DEL ESOFAGO

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En el 1900 Preble[1] informó 60 casos de hemorragia severa del trayecto gastrointestinal enfocando la atención sobre las varices sangrantes del esófago. En la serie de Welch[2] las varices sangrantes constituían la etiología en el 32% de las hemorragias gastrointestinales. En pacientes con cirrosis hepática éste porcentaje aumenta a 59.

Diagnóstico:

La identificación definitiva de las varices sangrandes como causa de la hemorragia, constituye una problema difícil. Casi todos los pacientes presentan un problema de urgencia muy serio.

La asociación de ulcus gástrico o duodenale, gastritis alcohólica y erosiones gástricas superficiales con cirrosis, es una fuente de confusión en el diagnóstico.

La evidencia clínica por lo general se enfoca hacia el diagnóstico de cirrosis soslayando el de varices.

Los hallazgos de laboratorio son de considerable valor. La prueba de bromosulfotaleína para función hepática siempre ha sido notablemente anormal en pacientes padeciendo de cirrosis, en contraste con la alteración mínima en aquellos pacientes con úlcera duodenal. Sin embargo es una prueba muy tediosa cuando afrontamos una situación de urgencia.

La protrombina del plasma está consistentemente disminuída en los cirróticos.

En la úlcera duodenal sangrante el tiempo de protrombina raras veces está prolongado inicialmente. Ocasionalmente se nota evidencia de hiperesplenismo tales como pancitopenia y trombocitopenia.

El estudio del esófago por medio del esofagograma no demuestra las varices consistentemente en aquellos casos que no sangran. Es mucho más difícil sin embargo demostrar las varices en aquellos casos que están sangrando activamente.

La esofagoscopía sería la mejor ayuda en el diagnóstico, pero frecuentemente la regurgitación de sangre del estómago obscurece el campo visual.

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Control de la Hemorragia

El choque existente exige la administración de transfusiones de sangre. La sangre fresca es preferible a la almacenada porque tiene un contenido mayor de protrombina y menor de amonia.

El tapenamiento[3,4,5] pneumático por lo general controla la hemorragia de las varices esofágicas pero puede fallar en dominar la hemorragia de una várice sangrante localizada en el fondo gástrico.

El taponamiento efectivo se manifiesta dentro de las primeras seis a doce horas y no debe prolongarse por más de veinticuatro, debido al posible edema e inflamación del área afectada. Si la hemorragia reaparece al desinflar el balón superior después de un control inicial, la intervención quirúrgica debe ser considerada seriamente.

Intervenciones quirúrgicas para dominar la hemorragia aguda de las varices.

1. La esplenectomía[6] fué el primer procedimiento usado. Los resultados fueron de gran éxito en un limitado grupo de pacientes, en los cuales la oclusión segmentaria de la vena esplénica era exclusivamente responsable por la hipertensión portal extrahepática. En el cirrótico este procedimiento falla.

2. La ligadura de las venas coronarias también ha fallado. La ligadura de la arteria esplénica fué introducida por Blain[7] en 1918. Más recientemente Rienhoff[8] y Boerman[9] han propuesto la ligadura de las arterias hepática, gástrica y esplénica.

Los resultados clínicos han sido muy variados. El informe de Madden[9] ha aumentado el escepticismo en cuanto al valor de este método. Som y Garlock[10] en 1947 introdujeron el tapenamiento del mediastino que consiste en hacer un empaquetamiento alrededor del esófago. Este método no ha sido aceptado generalmente.

La resección esofago-gástrica[11] también se ha intentado pero la escasez de los resultados informados hasta la fecha nos obligan a pensar que pocos pacientes han sido seleccionados para un procedimiento tan formidable ya que los resultados no han sido consistentemente buenos. Cole ha dicho que la transacción del cardias con ligadura de venas, es preferible a una portocava de urgencia.

Las portocavas se han efectuado en situaciones de urgencia pero aparte del éxito informado por Child[12] los resultados han sido generalmente pobres.

La ligadura transesofágica de las várices fué propuesta por Boerema[13] en Holanda en 1949. Crile[14] y Linton[15] adoptaron este método.

Nosotros hemos hecho ligaduras transesofágicas en cuatro casos de várices sangrantes. Todos han sido verdaderos problemas de urgencia en los cuales los procedimientos conservadores ya habían fracasado. Debido a la pobre condición de estos pacientes y por la magnitud de la intervención podemos considerar estas intervenciones como verdaderamente heroicas. Sin embargo todos los pacientes las toleraron muy satisfactoriamente.

Técnica Quirúrgica:

Usando anestesia endotraqueal con paciente en decúbito lateral derecho y manteniendo el tubo de Sengstaken[5] inflado, hacemos una toracotomía izquierda, entrando en el hemitórax izquierdo, a través del séptimo espacio intercostal. Retraemos el pulmón anteriormente y abrimos la pleura en el mediastino posterior. Identificamos el esófago y lo levantamos de su lecho. Lo separamos de la abertura diafragmática y dividimos el diafragma. Liberamos la región del cardias para mobilizar el estómago. Una vez que el esófago distal, el cardias y el fondo gástrico están libres, ordenamos la desinflación del tubo y entonces dividimos el esófago longitudinalmente sin incluir el cardia. Abrimos también el fondo gástrico. Identificamos las várices sangrantes y le colocamos múltiples ligaduras de transfixión. Cerramos el esófago y el estómago dejando un tubo de Levin después de sacar el tubo de Sengstaken. Reparamos el diafragma y cerramos el hemitorax con drenaje bajo agua.

El tratamiento post operatorio es el usado usualmente en la cirugía esofágica.

Presentación de Casos:

M. C. mujer de 48 años de edad, fué admitida al Hospital de Distrito de Ponce el día 2 de enero de 1957 con hematemesis profusa. Había sangrado el 23 de diciembre de 1956 y el primero de enero de 1957. Varios años antes había sido sometida a una esplenectomía.

El exámen físico demostraba una mujer blanca, gruesa, con hígado tres traveses de dedo por debajo del reborde costal. Corazón y pulmones negativos. Presión arterial 60 40. Se le administraron 1000 cc de sangre. Se colocó el tubo de Sengstaken y se mantuvo inflado casi por 48 horas. Al desinflarlo, la paciente volvió a sangrar y entonces decidimos intervenir. La hemoglobina era de 56 y el contaje de hematíes 2,510,000. Teniendo 1500 cc de sangre disponibles practicamos la operación. Encontramos várices enormes que tenían hasta 1.5 cm. de diámetro. Una de ellas más

impresionante que las demás, estaba localizada en el fondo gástrico y sangraba exageradamente con una presión casi arterial. Hicimos ligaduras múltiples y paramos la hemorragia. El post operatorio se complicó con una fístula pleuroesofágica que cerró con tratamiento conservador. Después de haber recobrado de esta intervención le propusimos una anastomosis portocaval y la paciente no la aceptó. Fué dada de alta el 9 de febrero de 1957.

J. A., varón de 62 años fué admitido al Hospital de Distrito el 16 de marzo de 1957 con hematemesis. Hacía una semana que había sido dado de alta de otro hospital donde había estado con hematemesis también. Durante los dos meses anteriores había tenido varios episodios de hemorragia por la boca.

El exámen físico reveló un hígado palpable dos traveses de dedo por debajo del reborde costal. El bazo no era palpable. Tenía sensibilidad marcada en el epigastrio. El corazón y los pulmones no mostraban ninguna anormalidad. La presión arterial era de 110/80. La hemoglobina era 48% y el contaje de hematíes era 3,090,000.

Recibió varias transfusiones de sangre y dejó de sangrar espontáneamente. El 25 de marzo se le hizo un esofagograma, pero no se pudieron visualizar las várices. El día 3 de abril una biopsia rectal resultó positiva para Schistosoma Mansoni.

El día 8 de abril ocurrió otro episodio de hematemesis. Esta vez se le colocó el tubo Sengstaken que se mantuvo hasta el décimo día. Después de múltiples transfusiones de sangre subió la hemoglobina a 68% (09.86 gr.) y los hematíes a 3,880,000. Fué llevado a la sala de operaciones. En la operación se encontraron várices esofágicas enormes, y se practicaron múltiples ligaduras de las mismas. La intervención fué muy bien tolerada. Después de haber convalecido de esta operación el día 30 de junio se le practicó una anastomosis portocaval, habiendo recobrado satisfactoriamente. Fué dado de alta el 14 de julio de 1957 en buenas condiciones.

J. M. F., varón de 61 años, fué admitido al hospital de Distrito el 6 de julio de 1957, después de haber sufrido un episodio de hematemesis. Nunca había sangrado antes. El paciente se veía muy pálido y su apariencia física era muy pobre.

El hígado era palpable a dos traveses de dedo por debajo del reborde costal derecho mientras se observaba el bazo también a igual nivel. El corazón demostraba un soplo mitral grado I. Los pulmones eran negativos. La presión arterial 110/60. El hemograma reveló 2,070,000 hematíes y 5 gm. de hemoglobina. El día 7 de julio se hizo un esofagograma y las várices pudieron demostrarse. Se le dieron 500 cc de sangre el 8 de julio. No se consiguió más sangre hasta el 24 de julio cuando se le administraron otros 500 cc. Se trató de conseguir suficiente sangre para someter el paciente

a una portocava. Pasado algun tiempo, nuevamente, el día 8 de septiembre tuvo un nuevo episcdio de hemorragia. Se le insertó un tubo de Sengstaken y la hemorragia fué corregida. Esta vez se consiguió sangre suficiente y el día 10 de septiembre se hicieron múltiples ligaduras de las várices. El paciente tuvo enterorragia inmediatamente después de la operación, ignorándose el origen del nuevo episodio. La enterorragia cesó y el paciente fué mejorando gradualmente.

Fué dado de alta el 4 de octubre de 1957 para que consiguiera sangre para ser sometido al procedimiento definitivo.

L.R.P., varón de 43 años fué admitido el 4 de octubre de 1957 al Hospital de Distrito de Ponce con una hematemesis profusa. Había sido tratado hacía 15 años para Bilharzia. Tenía historia de alcoholismo y se quejaba de dolor en el epigastrio.

Al examen físico los pulmones y el corazón eran negativos. No había sensibilidad en el abdomen. El hígado era palpable a tres traveses de dedo por debajo del reborde costal derecho y el bazo a dos por debajo del reborde izquierdo. Al ser admitido la hemoglobina indicaba un índice de 34%, (5 gm.) y el hematocrito de 18%. La protrombina era de 18 segundos con un control de catorce segundos. Se colocó el tubo de Sengstaken y la hemorragia cesó.

El día seis se desinfló el tubo y el paciente sangró nuevamente. El día siete fué operado. El paciente fué conducido a la sala de operación con un 60%, (8.7 gr.) de hemoglobina, 2,500,000 hematíes y un hematocrito de 25%.

Se encontraron várices enormes en el tercio inferior del esófago y en el fondo gástrico. Se hicieron múltiples ligaduras de las várices, habiéndose inyectado 2000 cc de sangre. No hubo complicaciones y fué dado de alta el 27 de noviembre con la recomendación de conseguir sangre para una portocava.

RESUMEN

1. Hemos analizado los diferentes procedimientos a nuestra disposición con miras a resolver el problema de la hemorragia activa en los casos de várices sangrantes del esófago.

2. Hemos presentado cuatro pacientes con várices sangrantes en los cuales se practicaron ligaduras múltiples con éxito.

3. Creemos que este último procedimiento es el más efectivo y de menos riesgo.

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THE SURGICAL TREATMENT OF ORAL CANCER

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In the discussion of oral cancer one cannot avoid the ever conflicting claims of the radiotherapist and of the surgeons in this field. Because of the very poor results with surgery in the beginning, it was almost invariably the custom to surrender the cases of mouth cancer to the radiotherapist, so that even at the start, they had long since displayed their surgical colleagues. As time went on, the surgical potential of cure of oral cancer has been enhanced to such a degree that it deserves a review by all those antagonistic to this form of treatment. In the late 30's - 40's several important surgical advances were made which eliminated many of the hazards which had deterred surgery of the head and neck from progress. Surgery has evolved a great deal from the first approaches by the transoral local excision of lesions, which were often combined with subsequent sepsis, slough, prolonged and painful healing; and with such poor cure rates except in the very superficial and small lesions, which made if fall into disrepute. It was the occasional cure by irradiation of the very extensive and often inoperable lesion which made this the treatment of choice. With improvement in X-ray dosages one was able to obtain longer survivals; at the same time, radionecrosis became a factor in the cured cases, many of which required later operations for removal of necrotic, infected bone; so much so, that sequestrectomy became a radiation by-product in a certain number of patients. As time went on, it was found that cases with bone involvement did not respond well to X-ray and that they were better treated by surgery. It was also found that one of the main factors in determining curability was the presence of neck metastases; a condition which also pointed to surgery as the treatment of choice. Thus, the operation of block dissection was devised. It was the late George W. Crile who first realized that for the treatment of cervical metastases, an en-block resection was necessary. He was able to prove that the salvage of metastatic neck cancer, although still poor at that early period, was much better with surgery than with radiotherapy. He often combined neck dissection with the operation of a primary lesion in the mouth, but he had such a prohibitive mortality that these operations were done less and less frequently.

At Memorial Hospital up to 1930, mandibular resection were mainly limited to cases of necrosis or osteomyelitis, and only occasionally for primary tumors of bone. These were done thru the

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oral route to prevent salivary fistulas which frequently occurred with the external approach. With the advent of sulfa drugs in the mid 30's they could be performed thru the sub-mandibular route with primary healing. Sometimes it was combined with neck dissection; until finally in 1938, the now practiced combined resection was finally instituted. With continued success, the operation was gradually extended to lesions of the floor of the mouth, cheek mucosa, tongue, including the base, tonsillar area, and still later to lesions of the pharynx and larynx.

The factors which influenced the progress in the surgical treatment were: first, the perfection of endotracheal anesthesia, with its attendant safety in prolonged, traumatizing operations; whereby, the airway was completely sealed off from the operative field; blood and secretions presenting no problem to the operating surgeon, who could now operate on a patient in a light plane of anesthesia for several hours, completely under control at all times, and practically awake and reacting at the completion of the operation. Secondly, the antibiotics have practically eliminated wound infections in spite of the wide open and grossly contaminated field thus reducing the high mortality due to uncontrolled infection. Thirdly, blood banks with almost unlimited amounts of blood available for replacement when necessary or desired coupled with our knowledge of prevention and treatment of shock. Another factor is our knowledge of the repair of nutritionally depleted patients, which make up the great majority of our cases, specially those radiation failures with persistant or recurrent disease. Many are in such poor state of nutrition and metabolic depletion that they constitute an almost impossible surgical risk. Quite often, in a short period of time, these patients can be restored to a fairly normal state and are able to withstand prolonged surgical interventions with minimal risks.

With all these advances surgery has taken a gigantic step forward and this, coupled with the natural history of epidermoid carcinoma, which metastasizes practically always thru the lymphatics, sparing the venous channels, has evoked the rationale for on operation which might cure cancer far better than other types of treatment. This constitutes the excision of the primary lesion, be it in the tongue, floor of the mouth, gums or cheek mucosa, coupled with a ressection in continuity of all the lymphatic draining areas of the neck. We have observed time and again, patients who died of head and neck cancer, who have admittedly advanced disease, and who very seldom show evidence of spread below the clavicles. It is essentially a regionally localized disease, that kills thru the effects of local destruction, obstruction of the airway, interferences with nutrition, and erosion of the main vessels. We

have so arrived at our present state in the surgical treatment of cancer of the oral cavity, where the limits of operability are determined mostly by the necessity of preserving structures which are essential for life, and not by those of former years; namely, blood loss, shock, asphyxia, uncontrollable infection, and prolonged morbidity and poor healing. We should not, however, let our enthusiasm get the best of mature judgement, and in spite of the fact that most tumors of the head and neck can be removed by the experienced surgeon, there is a limit to what radical surgery can accomplish.

I have reviewed our material at Dr. I. González Martínez Oncologic Hospital, limiting myself to cancer of the gums, oral mucosa and floor of the mouth. 160 cases have been compiled of which 66 are of cancer of the gums, 42 of the cheek mucosa and 52 of the floor of the mouth. The age distribution can be seen in Tables I to III where the greater number of cases are found in the 7th decade. Exception is made in the case of floor of the mouth cancer where the 6th decade has the greater number of cases. May I point out the rather unusual finding of a case only 17 years old.

Table I

CARCINOMA OF GUMS

Age Incidence

30-40	7		
40-50	10		
50-60	14	Males	42
60-70	24	Females	24
70-80	7		
80-90	4		

Table II

CARCINOMA OF ORAL MUCOSA

Age Incidence

30-40	1
40-50	3
50-60	11
60-70	19
70-80	4
80-90	4
TOTAL	42

Table III
CARCINOMA OF FLOOR OF MOUTH

Age Incidence

10-20	1
30-40	1
40-50	10
50-60	17
60-70	13
70-80	9
80-90	1

Oral cancer is fairly common in Puerto Rico occupying 14% of male carcinomas. Factors of importance in its etiology are shown in Tables IV and V. There is a high incidence of the use of tobacco either by smoking or by chewing. In the case of oral mucosae lesions it amounts to 71.4% ; available data does not allow us to determine exactly how many of these chewed or smoked, although a good number were chewers. Another rather frequent finding is that of a poor denture, which here amounts to 59.5%. Lues, which has often been referred to as a predisposing cause was only present in 9.5% of cases. There is no variance in sex incidence. In the case of floor of the mouth lesions tobacco users

Table IV
CARCINOMA OF FLOOR OF MOUTH

Etiological Factors

Tobacco	41	78.8%
Lues	8	15.3%
Poor denture	35	67.3%
Males	44	84.6%
Famales	8	15.4%

Table V
CARCINOMA OF ORAL MUCOSA

Etiological Factors

Tobacco	30	71.4%
Lues	4	9.5%
Poor denture	25	59.5%
Males	21	
Females	21	
TOTAL CASES	42	

are more plentiful accounting for 78.8%. Here again, poor dentures, with pyorrhea, jagged, dirty and infected roots, is quite prominent — 67.3%. Contrary to cancer of the gums the males far outnumber females, almost sixfold, with 84.6%. This finding is compatible with that reported by other investigators who find the incidence in males is about 6 to 8 times that of females.

The type of treatment has varied a great deal throughout the years; and in our Institution, which started almost exclusively as a radiotherapy center, it was a logical corollary that the majority of cases were treated by irradiation specially in the earlier years. Table VI shows the type of treatment utilized in the oral mucosa group. Three cases were too advanced and no treatment was given. The rest of the cases show the prevalence of irradiation over surgery; almost two thirds of the cases being treated with either x-ray or in combination with radium. About the same situation prevails in the floor of the mouth lesions (Table VII) where x-ray and radium were used in over half of the cases. Table VIII shows the same number of cases of cancer of the gums treated surgically as by irradiation.

Table VI
CARCINOMA OF ORAL MUCOSA

Type of Treatment

Not treated	3
Local excision	3
Radium & X-ray	8
Radical Operation	11
X-ray	17
TOTAL CASES	42

Table VII
CARCINOMA OF FLOOR OF MOUTH

Type of Treatment

Not treated	2
Local excision + Irradiation	5
Radium & X-ray	12
X-ray	16
Radical Surgery	17
TOTAL	52

Table VIII
CARCINOMA OF GUMS
Type of Treatment

Not Treated	2
Local excision	3
Radium & X-ray	4
Primary Surgery	7
Post X-ray	23
X-ray	27
TOTAL	66

An important part of our study was determining the relative results with the different methods of treatment. It may be rather arbitrary to postulate that the results definitely indicate superiority of one method over another; many other factors would have to be considered such as the stage of the disease and the applicability of one or the other method. Table IX shows our results in cancer of the gums. The best results were obtained with local excision, being able to control the disease in 2 out of 3 cases. This does not mean, of course, that it is the best treatment; we were just fortunate in getting three early and localized cases which could be treated favorably by this limited method. It is in these cases where the poor results obtained with irradiation are more vividly demonstrated being able to control only 11.1% of cases. It is important to note that 27 cases received x-ray solely as a method of treatment, but that the 23 cases operated on secondarily had already received x-ray and had either failed to be controlled or had recurrent disease. Thus, out of 54 cases receiving irradiation, 51 cases were actual failures. One of the failures with radium and x-ray had been actually controlled for about 5 years, later to develop uncontrollable recurrence dying 2 years later. Of the 23 cases in which irradiation had failed, and who were operated on, we were able to salvage 10 or 43.4%. It is in this group that we have two of our older survivals — 11 year free of disease. In the cases treated primarily by surgery we met our Waterloo; the unfortunate occurrence of two deaths from shock immediately after operation and another of a coronary thrombosis on her six postoperative day. In spite of this we were able to salvage 42.8%, which is certainly well above the curability obtained by irradiation. It approaches one of the best reported in the U.S.A. by Modlin and Johnson[10] who were able to obtain 47% in 5 years survival, with a 6% mortality. Our over all salvage of 27.2% compares favorably with other figures reported which vary between 28-29%.

Table IX
CARCINOMA OF GUMS

Results of Treatment

		Failures	Controlled	%
Not treated	2	2		
Local excision	3	1	2	66.6%
Radium & X-ray	4	4		
Radical Surgery				
Primary	7	4	3	42.8%
Post X-ray	23	13	10	43.4%
X-Ray	27	24	3	11.1%
Total	66	24	3	11.1%
Operative Mortality		12%		

In Table X, cancer of the oral mucosa also shows poor results with X-ray, 17.6%, although in combination with radium the results were improved to 37.5%. However, with radical operation we were able to salvage 7 out of 11 cases, totalling 63.6%. Our total salvage for this group was 30.9%.

Table X
CARCINOMA OF ORAL MUCOSA

Results of Treatment

		Failures	Controlled	%
Not treated	3	3		
Local excision				
+ X-ray	3	3		
Radium & X-ray	8	5	3	37.5%
Radical Op.	11	4	7	63.6%
X-ray	17	14	3	17.6%
Total	42	29	13	30.9%

In the last group of cancer of floor of the mouth, (Table XI) we also find interesting points. Local excision plus irradiation which was limited to small and early lesions, gave a rather good result — controlling 2 out of 5 cases. The cases treated by irradiation, either X-ray of X-ray plus radium, still leave much to be desired as far as results, only salvaging 31.2%. Here too we have obtained better results with surgery controlling 7 out of 17, 41.1%. We had one operative death, which outopsy proved to be due to obs-

truction and atelectasis from a left main bronchus cancer which the preoperative X-ray failed to show. This mortality of 5.8% was figured erroneously, since the cases of local excision were not considered. Actually mortality is 4.5%. If we take all the cases treated surgically our mortality is only 5.7%. Our overall salvage was 25%, comparing favorably with the results reported by Sir Stanford Cade,[1] Gordham,[2] and Dobbie[3] who have between 26.7% and 29%. Slaughter[5] reports 32% in 120 cases of combined res-section, but he states that at first only the cases which had failed with irradiation were referred for surgical treatment; and only lately have more cases been treated primarily by surgery with better results.

Table XI
CARCINOMA OF FLOOR OF MOUTH
Results of Treatment

		Failures	Controlled	%
Not treated	2	2		
Local Excision + Irradiation	5	2	2	40%
Radium & X-ray	12	9	3	25%
X-ray	16	15	1	6.2%
Radical Surgery	17	10	7	41.1%
TOTAL	52	39	13	25%
Operative Mortality		5.8%		

After reviewing our material it is with candor and sincerity that I say I am convinced that surgery has a definite place in the treatment of oral cancer, and that we can accomplish probably better results than those that can be accomplished in a comparable group of cases treated by irradiation. I am not in any way trying to imply that radiation has no place in the treatment of head and neck cancer. On the contrary, it is one of our most powerful weapons in our fight against this disease. I do believe, however, that more cases similar to those that had been treated by irradiation in years past should be given the oportunity of surgical treatment, and in the future we might be able to improve our results.

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EDITORIAL

AFUERA LOS MERCADERES

La profesión médica posee un código de ética cuyos dictados son claros, concisos e inspiradores. Este código es lo suficientemente elástico para cubrir situaciones imprevistas. Al mismo tiempo el código de ética profesional es lo suficientemente inflexible para no tolerar interpretaciones ficticias del mismo que convengan a intereses personales.

Si nos detuviéramos por un momento a examinar las actividades de nuestra profesión a la luz del código con toda probabilidad nos sorprenderíamos. En nuestra opinión la profesión médica en forma global observa los principios establecidos sólo a grandes rasgos. Si individualizáramos, como es natural, encontraríamos compañeros que lo obedecen religiosamente, otros que lo ignoran en su totalidad y una serie de matices de mayor a menor intensidad entre ambos extremos.

Todos los médicos estamos conscientes de los innumerables problemas que nos asedian diariamente como individuos y como grupo. Estos problemas atañen a los fundamentos más básicos de la profesión puesto que conciernen a la relación entre médico y paciente.

Somos de opinión que si escudriñamos muchos de estos problemas eliminando tecnicismos y datos superfluos encontraríamos violaciones al código de ética con bastante frecuencia, como móvil a la estructuración de estos problemas. En la misma forma estamos seguros que la corrección de esta situación ayudaría considerablemente a la solución de los mismos.

En la violación de los principios de ética el médico puede estar actuando como una unidad aislada que decide erróneamente sobre los cánones de conducta a seguir. En este caso compete a la sociedad médica el tratar de enmendarlo y finalmente castigarlo si fuese necesario. No es nuestro propósito evaluar la eficacia con que esto se está llevando a cabo, pero bien convendría a la organización correspondiente el hacerlo.

El médico que viola los principios de ética, sin embargo, puede estar actuando como una pieza integral en el mecanismo de una agrupación profesional, organización u hospital. Dentro de este mecanismo él puede abrazarse a cánones de conducta errónea por decisión propia o por lo que en la actualidad más nos preocupa, por presión o influencia del grupo con quien se asocia. Resulta muy doloroso pero tenemos que admitirlo que esta presión malsana a menudo se ejerce sobre individuos que por su juventud profesional, sus medios económicos limitados o ambas razones se sien-

ten obligados a ceder ante la amenaza ya sutil o franca de que pueden ser substituídos. Un ejemplo en forma de mueca amarga de las libertades unilaterales.

Aun si por el momento nos olvidamos del médico como individuo, no podemos olvidar el efecto detrimental que esta situación conlleva a la prestación de los mejores servicios médicos. Los principios de ética médica se fundamentan en la libertad profesional del médico y en la libertad del paciente para obtener los servicios. De esta libertad surge el esfuerzo máximo del médico desplegando todos los conocimientos a su haber en pro del enfermo. De esta libertad surge la remuneración adecuada del médico eficiente y proficiente en libre competencia en su ambiente.

Si nos tomamos el tiempo para revisar brevemente los principios de ética médica como han sido publicados por la Asociación Médica Americana y aceptados por la Asociación Médica de Puerto Rico nos encontramos con que los siguientes cánones son violentados con inquietante frecuencia dentro del marco de las instituciones hospitalarias y agrupaciones profesionales:
A saber:

1. Sección 5, Capítulo 7: Esta sección conlleva el que ningún médico rinda sus actividades profesionales a hospitales, grupos, organizaciones o individuos bajo condiciones que permiten la explotación del médico para beneficio económico de la otra parte. Estas actividades se catalogan como indignas y dañinas hacia la profesión y la comunidad.
2. Sección 4, Capítulo 7: Esta sección define la libertad que tiene todo paciente para escoger al médico que prefiere. En esta relación de médico y paciente una tercera persona o parte sólo es aceptada cuando asume responsabilidad legal y provee los gastos envueltos e indemnización por disabilidad ocupacional.
3. Sección 3, Capítulo 7: Esta sección define la práctica de la medicina por contrato. Aunque este tipo de medicina es aceptado, se le condena tan pronto conduce a que se deteriore la calidad de los servicios prestados. Es decir que las medidas administrativas que aumentan las ganancias negando servicios necesarios a pacientes de contrato no sólo nos son repulsivas, sino que ilegales y condenables por la ética profesional.
4. Sección 2, Capítulo 7: Esta sección prohibe que el médico preste servicios bajo condiciones que no le permitan rendir un servicio adecuado, excepto en circunstancias en que el paciente pudiera ser afectado adversamente, si no recibiera dicho cuidado. La última parte de esta sección, interpretada para la conveniencia de algunos, permite que esta

salvedad se perpetúe en perjuicio del fundamento de la sección y del paciente. Instituciones gubernamentales y al mismo tiempo varias privadas, son culpables de esta grave infracción sin propósito de enmienda.

5. Sección 6, Capítulo 2: Esta sección describe en detalle y sin ambigüedades el cobro de honorarios por servicios médicos. El violar esta sección del código de ética es una práctica más generalizada que lo que realizamos. No puede aceptarse que un médico pague a un individuo o institución por el privilegio de usar sus conocimientos y habilidad en beneficio de un paciente. El privilegio es del médico ya que sólo él u otro similar a él pueden rendir tal servicio. Esta práctica no puede ser condenada en tonos suficientemente enérgicos. Es cuna de toda clase de inmoralidades. Las maneras hábiles o relativamente hábiles con las cuales se tienden a ocultar estas prácticas indeseables de devolver cierta parte de los honorarios al "soi dissant" auto-agente-procurador o la práctica de permitir se retenga una parte de tales honorarios, deben ser perseguidas. Se nos ocurre, que existe una similaridad con el sistema de prostitución organizada.
6. Sección 4, Capítulo 1: Esta sección prohibe el que médicos, grupos profesionales, instituciones y organizaciones soliciten pacientes. Particularmente prohibe, el que se inciten comentarios a ser publicados en relación a médicos e instituciones en sus relaciones con determinados casos. Cataloga el elogiarse a sí mismo, como contrario a la moral y al buen gusto. Esta sección nos recuerda innumerables artículos y anuncios que han aparecido en nuestra prensa diaria y que jamás han debido publicarse.
7. Sección 3, Capítulo 1: Esta sección nos conduce de nuevo a nuestra línea original de pensamiento de la cual no debemos apartarnos. En síntesis nos recuerda que los principios de ética médica se aplican por igual al médico individual como al grupo, clínica o médico que proporcione empleo. Apunta con toda claridad que el que un médico entre en funciones de negocio u organizaciones profesionales no lo libera de las obligaciones éticas que asumió cuando ingresó en la profesión médica.

Esta sección es muy importante. En nuestro medio ambiente por varios años se ha aceptado, o al menos tolerado, el concepto de la personalidad dual del médico dueño de la institución, director de grupo profesional o director profesional de institución de propietarios laicos. Dentro de las actividades que conciernen el cuidado de enfermos esta doble personalidad es inadmisible ya que el código de ética profesional es monostótico y no acepta dua-

lidad en los cánones de conducta. Estos individuos por necesidad tienen que acatar los conceptos del código en el ejercicio de ambas funciones y descansa sobre la sociedad médica así demandarlo.

En nuestro medio ambiente la mayor parte de las facilidades a través de las cuales se prestan servicios médico-quirúrgicos y hospitalarios de índole privada son propiedad parcial o íntegra de médicos. Las otras facilidades de índole similar están bajo la dirección profesional también de médicos. Estos médicos por lo anteriormente expuesto no tienen otra alternativa, en aquellos casos que no lo estén haciendo, que ceñirse estrictamente a los principios de ética de la profesión. Los que fallaban en hacerlo parcialmente tienen que acatar la enmienda consiguiente.

Todas aquellas organizaciones que tengan dudas desde el punto de vista financiero sobre la observación de estos cánones debieran revisar seriamente si es que tienen razón alguna para subsistir como tal. La ineficiencia administrativa y el deseo de lucro incontrolable no son razones válidas para perpetuación.

Al terminar no deseamos entrar en consideraciones sobre si la sociedad médica puede o no imponer este código a aquellos que no lo acepten voluntariamente. Sería bien triste pensar que no pudiéramos dirigir los pasos de nuestra profesión hacia las cumbres, desviándola en todo lo posible de los abismos donde la naturaleza humana se satura del interés personal desmedido. Debemos evitar que el médico se aparte del código de ética y tratar de encauzarlo cuando fuese necesario. Debemos evitar que el médico sea indebidamente influenciado por grupos institucionales. Los mercaderes de salud no deben ser tolerados en nuestras filas.

brighten the day



for the moody patient...

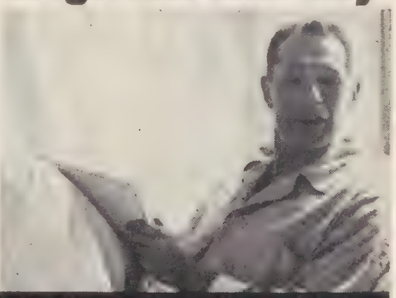
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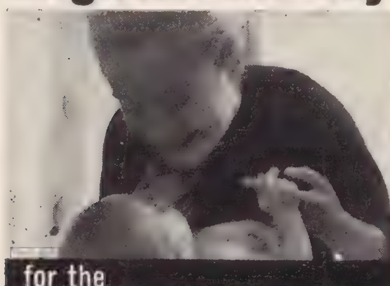
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for the depressed postpartum patient

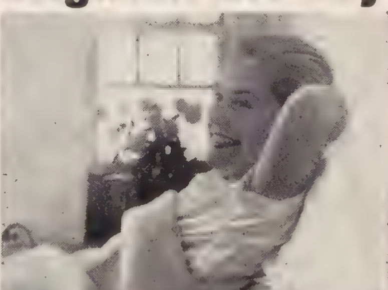
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